RESEARCH REVIEW ON TRAUMATIC BRAIN INJURY IN THE CRIMINAL JUSTICE SYSTEM

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ABSTRACT

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The criminal justice system seeks to establish social structure, mitigate crime, and sanction those who break the law. The system is used to delineate the crime, mark the offender, and designate appropriate punishment to the offender. However, this system would only be fair if the offender has full control and comprehensibility over their actions, which oftentimes is not the case. Scientific evidence has determined how morality, free will, and the different facets of one's personality is to some extent dictated by the electric and chemical signals firing in our brain. It has also been determined how our capacity for free will decreases and decision-making skills become impaired when our brain is damaged, a downstream consequence of which might be criminal tendencies. If this were to happen, the criminal justice system would have to apportion the blame to both the individual and the biological factors that make up the foundation of their actions. Some cases of this happening have convoluted the concept of criminal liability and the role of the individual in committing the crime.

The rate of individuals with Traumatic Brain Injury (TBI), resulting from damage to the brain, is much higher in prison and jail populations than in the United State's general population. Given the high prevalence of TBI in incarcerated populations, it is important to address how these individuals are affected, charged, and treated. This thesis will attempt to deepen the reader's knowledge of why criminals with TBI act the way they do, how they might be influenced by symptoms of their brain injury, as well as how the criminal justice system can best approach indictment, support, and treatment for offenders with history of TBI.

Part One will provide a general concept of TBI by going over general terminology, definition, and symptoms before establishing TBI as a causative factor of violence. Part Two will analyze different case studies on individuals who underwent a change post-TBI geared more towards violence and discuss the extent of their culpability if they had committed a crime as well as the different ways they were impacted by their brain injury. Part Three will delve into the criminal justice system by going over consequences of TBI in correction facilitates, obstacles towards proper identification and rehabilitation of victims of TBI, as well as steps forward. Part Four will delineate basic criminal proceedings, legal paradigms for the mentally affected, use of neuroscientific evidence in criminal trials, and lastly, establish standards to divide the responsible from the non-responsible.
The issue of how to approach offenders with brain damage is a difficult one, given how a clear casual relationship between neurological trauma and criminality has yet to be established.

However, hopefully by the end of this thesis, the reader will recognize a definite correlation between the two and have a better understanding of the moral, physical, and legal issues surrounding criminals with TBI.

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PART ONE: BACKGROUND

Overview and Definition of TBI

There are two major sub-sections of brain injury- congenital and acquired (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Congenital Brain Injuries usually result from a wide range of things that occur in utero before birth, such as encepathology, that affect the structural or physiological nature of the brain (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). It could also result from birth trauma or genetics (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Acquired Brain Injury is not hereditary, congenital, and degenerative but occurs outside of all these factors, and is divided into two categories- Traumatic and Non-Traumatic (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Non-Traumatic Brain Injuries are caused organically and can result from aneurysms, anoxia, encepathology, drug overdose, myocardial infarctions, etc…(Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Traumatic Brain Injuries (TBIs) are caused by an external force after birth and includes closed and open head injuries (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). During closed head injuries, the brain hits against the inside of the skull (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). During open head injuries, the brain is exposed and there could be something penetrating through the skull (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). This thesis will focus on solely on both open and closed TBI (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008).

There are three severities of TBI: Mild, Moderate, and Severe, and can be assigned to different brain injuries using the Glasgow Coma Scale (GCS) score, which could be accessible through medical records and are fairly indicative of whether or not the victim has had a brain

injury (Bridwell & MacDonald, 2014). The score ranges from 3 to 15, with 3 indicating that the user was completely unresponsive and 15 indicating that the user was completely responsive (Bridwell & MacDonald, 2014). A few different areas of the test include eye opening (1-4), motor response (1-6), and verbal response (1-5), and the summation of all the scores results in the final Glasgow Coma Scale score (Bridwell & MacDonald, 2014). However, the GCS may not be accurate in 100% of all cases (Bridwell & MacDonald, 2014). For example, although an individual who scored a very low 4 on the GCS probably has had a TBI in the severe category, everyone is different and there are abnormalities, such as a patient who had suffered through severe TBI but is completely functional and scores high on the GCS (Bridwell & MacDonald, 2014). Therefore, the different categories are also marked by other factors, like loss of consciousness and post-traumatic amnesia, which is a post-recovery process of the brain and marked by agitation, confusion, and loss of ability to make new memories (Bridwell & MacDonald, 2014). The longer PTA lasts, the more indicative an individual is not going to rehabilitate at a high level (Bridwell & MacDonald, 2014).

Mild TBI is defined by: Altered or Loss of Consciousness lasting less than 30 minutes with normal CT and/or and MRI GCS of 13-14 and Post-Traumatic Amnesia of less than 24 hours (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Moderate TBI is defined by: Loss of Consciousness lasting less than 6 hours with abnormal CT and/or MRI GCS of 9-12 and Post-Traumatic Amnesia of less than 7 days (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Lastly, Severe TBI is defined by: Loss of Consciousness lasting less than 6 hours with abnormal and/or MRI GCS of less than 9 and Post-Traumatic Amnesia of less than 7 days (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). Length of hospital stay could be a good indicator of severity of TBI as well (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008).

Majority (80-90%) of individuals with mild TBI recover, although repeated TBIs can result in loss of long-term functions (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). A victim who has sustained a TBI is twice more likely to sustain a second TBI (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). However, a "miserable minority" acquire chronic disability and prolonged, ongoing psychiatric symptoms (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008).

Table 1. Definition of mild, moderate, and severe TBI (Department of Defense & Department of Veterans Affairs, 2009)

Criteria	Mild	Moderate	Severe
Structural imaging (i.e., computed tomography; CT)	Normal	Normal or Abnormal	Normal or Abnormal
Loss of consciousness (LOC)	0-30 min	> 30 min and < 24 hrs	> 24 hrs
Alteration of consciousness ("dazed" feeling, confusion)	≤ 24 hrs	> 24 hrs	> 24 hrs
Post-traumatic amnesia (PTA)	≤ 24 hrs	> 24 hrs and < 7 days	> seven days
Acute Glasgow Coma Scale (GCS)	Score 13-15	Score 9-12	Score 3-8

The official definition of TBI is "alteration in brain function, or other evidence of brain pathology, caused by an external force" (Blyth & Bazarian, 2011). It is important to establish a clear definition of this term to interpret different case studies on TBI and also establish criteria for considering a diagnosis of TBI in the case of an individual on trial (Blyth & Bazarian, 2011). TBI is different from just a common head injury, which is less serious and limited to injury to the face and the scalp, whereas TBI is damage to the brain that can result from various injury mechanisms (Blyth & Bazarian, 2011). Individuals can be afflicted with mild, moderate, and severe TBI, and these three categories can be differentiated through their corresponding clinical

signs (Blyth & Bazarian, 2011). It's helpful to first break down the definition of TBI (Blyth & Bazarian, 2011).

[1] Alteration in Brain Function [can be defined by any of these symptoms]...:

a. Decreased or loss of consciousness (LOC)
b. Retrograde amnesia or post traumatic amnesia (PTA), in other words, incomplete memory for an event
c. Neurological deficits (weakness, aphasia, sensory loss, paralysis, blurry vision, etc…)
d. Change in mental state at the time of the injury (confusion, dull mental skills, etc…)

[2] ...or other evidence of brain pathology [that are usually examined using different imaging using sensitive biomarkers or lab techniques]

a. Clinical consequences as subtle or delayed
b. Clinical diagnosis is confounded by a difficult context
c. There is a need to differentiate TBI induced clinical signs from those with other causes

[3]...caused by an external force [may include any of the following]...

a. Object striking head, and vice versa
b. Brain undergoing rapid acceleration or deceleration
c. Head being penetrated by foreign object
d. Head exposed to force from blast, explosion, etc…

It was difficult for clinicians to define the criteria for Part [1] of the definition, as LOS, PTA, and other alterations of consciousness is typically gathered from the victim and cannot be accurately verified (Blyth & Bazarian, 2011). Thorough clinical interviews are conducted, especially when the injury occurs in the absence of witnesses, to reduce the uncertainty of diagnosis (Blyth & Bazarian, 2011). Usually, the occurrence of an alteration of consciousness, like confusion or nausea, without the presence of LOC or PTA is diagnosed as mild TBI, although it is hard to differentiate mild TBI from non-TBI pathology (Blyth & Bazarian, 2011).

Presence of PTA and alteration of consciousness typically occurs in TBIs resulting from contact sports (Blyth & Bazarian, 2011). However, TBI may be diagnosed when there is a clear injury to the brain, from maybe a small penetrating object, even without the presence of any symptoms (Blyth & Bazarian, 2011). Conversely, existence of the mental symptoms without clear brain injury should not serve as proof of TBI and may result from other causes, like medication, intoxication, drug use, etc...(Blyth & Bazarian, 2011) In addition, neuropsychiatric sequelae, including depression, impulsivity, and apathy, was deemed unfit as criteria to diagnosis TBI as it would constitute a circular argument (Blyth & Bazarian, 2011).

As can be seen from the Part [3] of the definition, TBI typically results from contact and inertial forces, both attributed to different biomechanical effects, acting on the brain (Blyth & Bazarian, 2011). This means that TBI can occur without the victim being in direct contact with any object (Blyth & Bazarian, 2011). Contact forces occur when the head of a victim strikes against an object with a great enough force to cause the brain to strike against the inner surface of the skull (Blyth & Bazarian, 2011). The brain striking against the ridges and bony protuberances of the frontal and temporal skull could potentially damage the temporal and frontal poles, and frontal, ventral cortices of the brain (Blyth & Bazarian, 2011). Inertial forces include linear translation and rotational forces that produce an angular change in speed and can result in shearing, compression, and straining of brain tissue (Blyth & Bazarian, 2011). When these forces overcome the integrity of the tissue, injury can result at the planes between different types of brain tissue, like junction between the gray and white matter, and at the brain stem, which holds the rotational center of mass (Blyth & Bazarian, 2011). These injuries can also impact axons of neurons, small blood vessels, the corpus callosum, and other fragile structures holding the brain together (Blyth & Bazarian, 2011).

Since the symptoms of TBI are usually non-specific, ambiguous, and likely to be confounded by non-TBI mechanisms, one option is to differentiate between degrees of precision for criteria that is used for "considering" and "establishing" the diagnosis of TBI (Blyth & Bazarian, 2011). Criteria used to "consider" diagnosis of TBI would include neurocognitive and neuropsychiatric sequelae that may have resulted from TBI, and the occurrence of injuries that don't completely meet the criteria in Part [1] of the definition (Blyth & Bazarian, 2011). Criteria used to "establish" diagnosis of TBI would have to be unambiguous, comprising of clear damage to the brain, and unlikely to be confounded by symptoms unrelated to TBI (Blyth & Bazarian, 2011). It is important to take into account "consideration" of TBI rather than dismiss it entirely as it may eliminate the chance to detect other causes and options of treatment (Blyth & Bazarian, 2011).

The injuries resulting from regular TBI can also be divided into 2 categories: Primary brain injury and secondary brain injury (Mckee & Daneshvar, 2015). Primary brain injury results from initial structural injury to the brain as a direct result of trauma, whereas secondary brain injury refers to subsequent injury, or a complication of, after initial injury (Mckee & Daneshvar, 2015). Secondary brain injury can occur after biochemical complications resulting from the initial injury, such as hypotension, hypoxia, and elevated intracranial pressure, and causes substantial damage to the brain (Mckee & Daneshvar, 2015). The injuries resulting from blast TBI, occurring from blasts, can be divided into three categories: Primary, Secondary, and Tertiary (Mckee & Daneshvar, 2015). Blasts account for 66% of combat injuries, 20% of US soldiers who had been sent to Iraq and Afghanistan return with mild TBI (Mckee & Daneshvar, 2015). Primary Blast Injuries occur when a direct injury from blast shock waves are sent through the skull (Mckee & Daneshvar, 2015). Secondary Blast Injuries occur with energized fragments

propelled by an explosion impact the head (Mckee & Daneshvar, 2015). Tertiary Blast Injuries occur when the victim is thrown by the blast and their head collides with other objects (Mckee & Daneshvar, 2015).

TBI is displayed through a range of physical, cognitive, and emotion deficits (Graham, Rivara, Ford, 2014). Some common physical effects of TBI include impaired mobility, impaired sensory experiences, seizure disorders, mental and physical fatigability, chronic pain, headaches, sleep disorders, and dizziness (Graham, Rivara, Ford, 2014). TBI victims who are adolescents are more prone to the onset of sleep disorders, since sleep is responsible in developing the structure and function of the adolescent brain (Graham, Rivara, Ford, 2014). Sleep disorders can result in impaired new learning and memory skills (Graham, Rivara, Ford, 2014). Some cognitive effects of TBI, which can be misinterpreted as disobedience on part of the victim, include reduction in abstract reasoning capacity, difficulty in attention, judgement, interest, and complex information processing, increased mental fatigue, difficulty with short term memory, initiation deficits, confusion, basic intellectual deficits, slowness in thinking and performance, etc...(Graham, Rivara, Ford, 2014). Emotional and behavior effects of TBI include difficulty controlling anger, poor impulse control, mood swings, low frustration tolerance, altered sense of humor, agitation, rage reactions, euphoria, hyper-sexuality or hypo-sexuality, childlike emotional reactions or behavior, etc...(Graham, Rivara, Ford, 2014).

Neurobiological Consequences of TBI

Traumatic Brain Injury, especially when untreated, can result in significant cognitive deficits in memory and learning, attention span, and executive functions. TBI is detrimental to the brain because it incites brain atrophy and calcification, the extent of which is directly proportional to the severity of the TBI (Mckee, Daneshvar, 2015). In moderate and severe TBI,

the hippocampus is more likely to atrophy, which is a significant contributing factor to cognitive decline (Mckee, Daneshvar, 2015). TBI can expedite chronic neurodegeneration and culminate in the onset of different neurodegenerative diseases, such as Alzheimer's disease, Parkinson's disease, and Chronic Traumatic Encephalopathy (Mckee, Daneshvar, 2015). In fact, some victims develop dementia-like symptoms after only receiving minor head injuries. A comprehensive understanding of the molecular mechanisms of TBI is advantageous when trying to minimize the impact of secondary injuries and formulating possible pharmacological therapies to prevent or reverse cognitive impairment following TBI. Although much more is known about the neurobiological consequences of TBI than before, this knowledge still needs to be translated into direct clinical care and treatment.

As mentioned before, TBI has two injury phases: Primary and Secondary. Primary brain injury occurs during the initial impact, which triggers a pathway of biochemical processes that result in secondary brain injury, a culmination of ischemic, inflammatory, and cytotoxic processes meshing together (Mckee, Daneshvar, 2015). Primary brain injury results from external mechanical forces, such as acceleration linear, rotational, blunt impact, and penetrative forces, and can be categorized as focal, an injury occurring in a specific location, and diffuse, an injury covering a wider area (Mckee, Daneshvar, 2015). Focal injuries tend to have a range of symptoms depending on the region of head impacted, whereas diffuse injuries tend to have more consistent symptoms (Mckee, Daneshvar, 2015). However, both types of injury usually result in immediate neurological damage and leads to secondary changes that produce neuronal death and dysfunction (Mckee, Daneshvar, 2015).

Secondary neurological damage is produced by a cascade of biochemical pathways triggered by primary injury. One cytotoxic pathway that is triggered during the initial impact

results in the excessive release of excitotoxins, such as glutamate and aspartate, that alters the permeability of the cell wall and allows for increased calcium and sodium influx and activation of calcineurin and calmodulin (Mckee, Daneshvar, 2015). This triggers increased potassium efflux in an attempt to restore the electrochemical potential. The ensuing ion imbalances leads to cell swelling, cell death, and destruction of axons (Mckee, Daneshvar, 2015). This neuronal loss has been proved to contribute to cognitive deficits following TBI (Mckee, Daneshvar, 2015).

The inflammatory response, usually lasting hours to days, to the initial impact also contributes to secondary brain damage through increased release of interleukins (IL). The initial impact activates astrocytes and microglia residents and secretes many proinflammatory cytokines that are neurotoxic (Mckee, Daneshvar, 2015). Cytokines Tumor necrosis factor (TNF), IL-10, and IL-6 increases greatly within hours of initial impact, and serves to activate caspases, triggering cell death (Mckee, Daneshvar, 2015). This neuroinflammation and chronic microglial activation has been shown to contribute to cognitive dysfunction later on, and could become a target for therapy (Mckee, Daneshvar, 2015). The suppression of these proteins via administration of medical compounds has been proved to decrease the long-term cognitive impact of TBI (Mckee, Daneshvar, 2015). However, these proteins also have neuroprotective functions and can induce cell growth factors that can protect nerve cells against damage, so it is still not clear to what extent medication could prove useful (Mckee, Daneshvar, 2015). Additionally, the inflammatory response is not contained in the brain and can lead to multiple organ dysfunction (Mckee, Daneshvar, 2015).

Intracranial hypertension and cerebral ischemia are two of the most detrimental secondary injury processes that needs to be addressed in the ICU to reduce irreversible brain damage (Haddad, Arabi, 2012). Cerebral ischemia, due to disturbance of cerebral circulation

from initial impact, leads to brain damage and is responsible for 90% of deaths following closed head injuries (Haddad, Arabi, 2012). This is a condition in which there is insufficient blood flow to the brain for metabolism, which leads to poor oxygen and nutrient supply to the brain and may result in cerebral infarction, ischemic stroke, or death of brain tissue (Haddad, Arabi, 2012). Elevated intracranial hypertension, which has a very high correlation with poor outcome, can be detected through symptoms such as headaches, nausea, and vomiting (Haddad, Arabi, 2012). The Cerebral Blood Flow-Targeted Therapy has successfully reduced jugular venous desaturation, but also led to systemic complications that outweighed the benefits (Haddad, Arabi, 2012).

Cell apoptosis, or programmed cell death, has also been identified as an important contributor to secondary brain injury and occurs within contusion sites in the acute post-traumatic period, as well as regions distant from the site of initial impact weeks after the injury (Mckee, Daneshvar, 2015). The death of cells, although it may serve a protective role, hinders recovery progression and exacerbates brain injury by disrupting synaptic plasticity, which is important in learning and memory, and other central nervous functions (Mckee, Daneshvar, 2015). Some pharmacologic strategies have been made and experimented on in animal models of TBI to reduce the extent of apoptotic cell death, but all failed to influence outcomes after being translated into the clinical setting (Mckee, Daneshvar, 2015).

There have been improved outcomes for patients who might previously have had poor outcomes due to recent advances in pharmacology strategies, but the definitive factors contributing to these improved outcomes remain ambiguous.

Personality, Affect, and Behavioral Changes

Many victims of traumatic brain injury exhibit long-term behavioral consequences in addition to cognitive sequelae, such as personality change, depression, anxiety, and other complications, like alterations in mood and behavior. As a result, these individuals often have difficulty maintaining social relationships, employment, and other societal roles (Fann, Hart, Schomer, 2009). Personality change, which will be examined first, due to TBI is persistent and results from the neurophysiological effects occurring during primary and secondary injury. Personality change, which is comorbid with other disorders, is a disorder of mood regulation and is a collection of clinically diagnosed symptoms (Wilde, et al., 2015). It is categorized into multiple subtypes, all encompassing different symptoms, such as apathy, aggression, paranoia, impaired social judgment, etc... (Wilde, et al., 2015).

There have been several clinical studies conducted on individuals who exhibited debilitating personality changes after diagnosis of traumatic brain injury, such as a study that profiled children and adolescents with TBI by using the Neuropsychiatric Rating Schedule to describe symptomatology associated with personality changes (Max, Robertson, Lansing, 2001). Their subjects consisted of 94 individuals between the ages of 6 and 14 who had been hospitalized after onset of TBI (Max, Robertson, Lansing, 2001). Each of these subjects participated in a retrospective study that consisted of a semi-structured interview, based on the Neuropsychiatric Rating schedule, designed to identify personality change symptoms and subtypes (Max, Robertson, Lansing, 2001). They discovered that 59% of the subjects diagnosed with severe TBI had a persistent personality change for an average of two years post-injury, while only 5% of the subjects diagnosed with moderate and mild TBI exhibited a persistent personality change (Max, Robertson, Lansing, 2001). Personality change was found to be correlated with severity of injury, LOC of over 100 hours, and reduced adaptive and intellectual

functioning, and is diagnosed based mostly on a deviation from normal behavior (Max, Robertson, Lansing, 2001). Other positively rated items on the Neuropsychiatric Rating Schedule, with personality change having the highest correlation, included affective instability (49%), disinhibited vocalization/verbalization (41%), outbursts of aggression or rage (38%), impaired social judgment (38%), marked shifts from normal mood to irritability (41%), and marked shifts from normal mood to depression (8%) (Max, Robertson, Lansing, 2001). The diagnostic criteria for personality change in the study considered that children do not have a fixed personality and thus personality change, and so the researchers categorized those diagnosed with personality change into 4 main personality change subtypes, including affective instability/labile subtype, aggressive subtype, impaired social judgment subtype, apathetic subtype, and suspicious/paranoid subtype (Max, Robertson, Lansing, 2001). The affective instability/labile subtype was the most common (50%), and was marked by dysregulation of emotions, which includes both heightened and reduced affect, as well as irritability (Max, Robertson, Lansing, 2001). Some subjects experienced magnified emotions, including "feeling meaner" and "more outgoing," while others experienced an absence of emotion, such as the subject who was "flat" and another who was "emotionless until she [had] a mood swing" (Max, Robertson, Lansing, 2001). The aggressive subtype (40%) was the second most common among the subjects and marked by aggressive acts that were impulsive and motivated by anger and minor frustrations (Max, Robertson, Lansing, 2001). Subjects in the impaired social judgment subtype (38%) were characterized with impulsivity and problems with social discourse (Max, Robertson, Lansing, 2001), subjects categorized in the apathetic subtype (14%) exhibited apathy, and subjects in the paranoid subtype (5%) had false suspicions of other individual having malicious intent towards them (Max, Robertson, Lansing, 2001). One other notable symptom

associated with personality change is perseveration, or repetition of the same statements and ideas. For example, one of the subjects believed she was pregnant and repeated this belief many times in conversation (Max, Robertson, Lansing, 2001).

The most prevalent psychiatric disorder TBI victims develop is Major Depressive Disorder, a disorder that majorly impacts health, productivity, and quality of life. It also has been shown to increase neuropsychological impairment and slow the rate of cognitive recovery (Fann, Hart, Schomer, 2009). Depression results from direct or secondary injury to the brain tissue, with the affected displaying significantly reduced left prefrontal gray matter volumes in the ventrolateral and dorsolateral regions (Jorge, Robinson, Moser, Tateno, Crespo-Facorro, Arndt, 2004). It is derived from neuropathological changes produced by TBI that deactivates lateral and dorsal prefrontal cortices and overly activates ventral limbic and paralimbic structures like the amygdala (Jorge, Robinson, Moser, Tateno, Crespo-Facorro, Arndt, 2004). Approximately 33 to 42% of victims develop it within the first year and 61% of victims develop it within the first 7 years (Fann, Hart, Schomer, 2009). Unlike personality change risk, increased risk for depression is common among severe, moderate, and mild TBI, and often comes with an increased risk of suicide, anxiety, and aggressive behavior (Fann, Hart, Schomer, 2009).

Aggression and anxiety, common symptoms of the instability/labile and aggressive subtypes of personality change, are also prominent ongoing consequences of many individuals suffering from TBI. Prevalence estimates of post-TBI aggression vary from 11 to 34%, and may be a symptom of a personality change, mood disorder, and delirium resulting from TBI (Rao, et al.). Prior studies indicate that TBI-related aggression is correlated with frontal lobe lesions, pre-TBI psychosocial functioning, and a history of alcohol and substance abuse (Rao, et al.). One study examined aggression in 67 subjects during the first three months post-TBI to determine

factors associated with post-TBI psychiatric disorders (Rao, et al.). The subjects were given two study evaluations administered by a neuropsychiatrist, with one assessing pre-TBI psychiatric and psychosocial problems and the other assessing the same problems post-TBI (Rao, et al.). They assessed severity verbal and physical aggression with the Overt Aggression Scale and rated the overall severity of aggression in these subjects with a weighted score, and they also assessed the severity of each subject's TBI with the Glasgow Coma Scale (Rao, et al.). Other factors they took into account included medical co-morbidity, family history of psychiatric illness, behavior and legal problems, and also conducted neuroimaging and cognitive tests (Rao, et al.). They found the prevalence of verbal aggression to be the highest in 28.4% of the subjects, with the two most common symptoms being angry shouts and vicious cursing with moderate threats of violence (Rao, et al.). Those marked with aggression post-TBI also had a higher chance of developing major depression post-TBI, impaired psychosocial functioning, and increased dependence on activities of daily living (Rao, et al.). Almost none of the subjects exhibited physical aggression (Rao, et al.). Individuals post-TBI also may feel anxious, some to the point of a panic attack, due to cognitive impairments, such as difficulty reasoning and concentrating, in situations requiring lots of information-processing. Anxiety is also comorbid with mood disorders, for example, 66% of individuals who develop major depressive disorder post-TBI also develop generalized anxiety disorder (Jorge, 2008).

It has been established that TBI can cause many cognitive and behavioral changes, but it is hazier whether TBI alone causes risk of alcohol and substance abuse in individuals who had no such problems during the onset of TBI. However, it has been proven in some studies that TBI can increase drug and alcohol use in individuals who had no history of abuse prior to injury. One study reported that increased rates of substance abuse and depression relative to community

controls, and another survey discovered that individuals with no history of substance abuse had a 4.5 odds ratio of substance abuse within the first year post-TBI (Bjork, Grant, 2009). However, although alcohol and drug abuse has been established as a problem in TBI victims, it is often a secondary consequence and coping strategy arising from psychiatric and depressive symptoms post-TBI. It is not only a risk factor for TBI, but also an impediment to recovery post-TBI, and a problem that should be considered when addressing TBI victims (Bjork, Grant, 2009).

Many of the behavioral consequences of TBI, including personality change, increased aggression and anxiety, as well as increased risk of alcohol and substance abuse, arise as psychiatric complications of injury and are forms of affective dysregulation. Many of these consequences are comorbid, such as depression and anxiety, aggression and anxiety, and depression and substance abuse. Symptoms of personality change, such as apathy, aggression, and irritability, is significantly associated with deficits in neuropsychological domains, including intellectual function, processing speed, divided memory, executive function, etc...(Wilde, et al., 2015). Individuals with TBI have difficulty comprehending negative emotions and thus have more trouble regulating it. A clear mechanism that traces injury to the brain to behavioral symptomatology has yet to be discovered, but the pattern of brain network damage, especially if a process regulating affect is altered, might lead to neurocognitive dysfunction and personality change (Wilde, et al., 2015). Neural and psychological mechanisms, and the resulting behavioral consequences, most be understood to develop treatment strategies. Clinical trials stimulating brain networks that regulate affect and inhibiting networks that generate affect could provide more information. Cognitive rehabilitation that targets processing speed, attention, and memory may enhance executive function, and behavioral therapy may help as well (Wilde, et al., 2015). Therapy and treatment techniques will be discussed later in the thesis.

Establishing a Correlation between Traumatic Brain Injuries and Violence

It has now been established that TBI compromises important neurological functions for self-regulation and social behavior, but it is important to also recognize how TBI-related cognitive and behavioral problems can lead to aggressive behavior and result in perpetration of violence. Aggressive behavior post-TBI can include explosive behavior set off by minimal provocation and can occur without warning, which explains how many individuals post-TBI may exhibit offending behavior (Silver, et al., 2005).

Dorothy Lewis, a psychiatrist researcher, was one of the first to establish traumatic brain injury as an etiology of violence and was involved in a research study that outlined the biopsychosocial characteristics of 18 juveniles who had been sentenced to death in Texas and discovered, amongst other factors, evidence of brain damage as a common denominator for 17 out of the 18 cases. This article also brought to light the failure of clinicians to obtain medical, educational, and family histories of the juveniles to be used during sentencing and was one of the first research studies that explored the correlation between brain abnormalities and violent behavior amongst juvenile offenders.

This studies' sample size consisted of 18 out of the total 26 male juveniles who had received death penalty in Texas for committing homicide (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Their first neurologic evaluations were requested only after their sentencing, and not for suspected neuropathology or psychopathology (Lewis, Yeager, Blake, Bard, Strenziok, 2004). All the subjects were 17 years of age at the time they committed to murders, had been on death row for 2 to 14 years, and 17 out of the subjects were either in lower middle or lower socioeconomic classes with one in middle socioeconomic class (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Evaluations were conducted by a psychiatrist and a neurologist at the

Texas Department of Corrections Livingston facility in a private area in the span of 3.5 hours (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Each subject received psychiatric, neurologic, neuropsychological, and education assessments (Lewis, Yeager, Blake, Bard, Strenziok, 2004). . This consisted of an interview loosely structured on the Bellevue Adolescent Interview Schedule and the Dissociative Interview Schedule, an examination of scars on the face, head, and body, examinations of neurologic and mental status, cranial nerve function, motor, sensory, and cerebellar function, and lastly, an assessment of frontal lobe deficits (Lewis, Yeager, Blake, Bard, Strenziok, 2004). . The frontal lobe deficit assessment included evaluation of visual fixation, smooth-pursuit eye movements, antisaccade eye movements, paratonia, and prefrontal release signs (Lewis, Yeager, Blake, Bard, Strenziok, 2004).

Other types of data can be found directly in the article, such as psychiatric data, but for the purposes of this topic, only histories of central nervous system trauma and frontal lobe functioning of the subjects will be presented thoroughly (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Many of the subjects had a medical history that indicated some form of abnormal brain development (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Two of the subjects were delivered by C-section, the third was born two months premature, the fourth was was born with respiratory distress syndrome, and the fifth was born with a cleft palate and lip which is indicative of CNS problems (Lewis, Yeager, Blake, Bard, Strenziok, 2004). In addition to 33% of the subjects born with compromised CNS functioning, all but one subject experienced several head injuries with a subsequent loss of consciousness (Lewis, Yeager, Blake, Bard, Strenziok, 2004). These injuries were acquired through car/bike/skateboard accidents, blows to the head with a bat, falls, etc…(Lewis, Yeager, Blake, Bard, Strenziok, 2004).

The most common neurological examination result of the 17 subjects who had a history of brain injury were signs of prefrontal cortical impairment (Lewis, Yeager, Blake, Bard, Strenziok, 2004). 5 subjects had one abnormal prefrontal finding, 3 had two abnormal prefrontal findings, 2 had three abnormal prefrontal findings, and 3 had four or more abnormal prefrontal findings (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Majority of the subjects had displayed significant impairment on the Iowa Gambling Task, a test that simulates real-life decision making used to test cognition and emotion, and had trouble modifying behavior to avoid negative feedback and anticipate future consequences (Lewis, Yeager, Blake, Bard, Strenziok, 2004). These subjects were unable to avoid selecting cards from disadvantageous decks and only 3 out of the 18 subjects chose significantly fewer cards from the disadvantageous decks by the end of the task (Lewis, Yeager, Blake, Bard, Strenziok, 2004). Majority of the subjects made decisions similar to the ones made by patients with a damaged orbitomedial prefrontal cortex (Lewis, Yeager, Blake, Bard, Strenziok, 2004).

In addition to birth and accidental brain damage as well as limited prefrontal cortex function, 12 of the subjects exhibited psychiatric ailments (Lewis, Yeager, Blake, Bard, Strenziok, 2004). 8 subjects displayed symptoms of early-onset bipolar disorder, and 4 of the subjects displayed symptoms of early-onset schizoaffective psychosis (Lewis, Yeager, Blake, Bard, Strenziok, 2004). The remaining 6 were either hypomanic, retarded, psychotic, dissociative, sleep disordered, or some combination thereof (Lewis, Yeager, Blake, Bard, Strenziok, 2004). It is unclear whether these results were consequences of prior TBI, or a factor that predisposed the subjects to TBI (Lewis, Yeager, Blake, Bard, Strenziok, 2004). In addition, all of the subjects came from violent families with parents who were to some extent mentally unstable, and all had experienced physical and emotional abuse (Lewis, Yeager, Blake, Bard,

Strenziok, 2004). However, past studies have shown that violent juveniles often have parents who are unable to provide such guidance and act in the child's best interests (Lewis, Yeager, Blake, Bard, Strenziok, 2004). This is why it is important to thoroughly evaluate the juvenile's psychiatric, neurologic, and environmental circumstances before the trial, which did not occur in this case (Lewis, Yeager, Blake, Bard, Strenziok, 2004).

This case study was also the first to yield these unexpectedly high results- with 17 out of the 18 subjects experiencing some type of brain injury (Lewis, Yeager, Blake, Bard, Strenziok, 2004). This case is different from most others in how it involves the indictment of violent juveniles, but very clearly demonstrates how brain injury is definitely a factor that contributes to violence and impulse control problems (Lewis, Yeager, Blake, Bard, Strenziok, 2004).

Recent birth cohort and data linkage studies in adolescents and adults, the most common demographic for offenders, indicate that TBI can lead to increased risk of crime. However, although many of these studies establish associations between TBI and crime, as in criminal populations have a higher incidence of TBI, there hasn't yet been a clear causal mechanism delineated (Williams, et al., 2018). This tenuous link between TBI and TBI-caused violence is one of the reasons why TBI-induced criminality, which will be discussed later, remains a controversial topic. However, different theories for the etiology for aggression and impulse control will be discussed in the next section in relation to several case studies.

PART TWO: CASE STUDIES

Anatomy of the Brain

The brain controls all functions of the body, interprets all senses, and is the essence of the mind and the soul. Some may even say that morality and free will are dictated by the firing of neurons in the brain, which subject us to our emotions and personalities, and in some cases, offending behavior. This section of the thesis will look at various case studies of individuals whose mental states had significantly impacted their criminal tendencies in preparation to analyze the concept of criminal responsibility and the extent of free will in criminal decision-making with offenders post-TBI.

The brain has three main parts, the cerebrum, the cerebellum, and the brainstem. The cerebrum is the largest part of the brain, consisting of right and left hemispheres as well as outer cortex layers of grey matter and underlying regions of white matter, and controls all voluntary actions in the body (Hines, 2018). It contains the cerebral cortex as well as subcortical structures, the hippocampus, basal ganglia, and olfactory bulb (Hines, 2018). The cerebral cortex is further divided into four main lobes: frontal, parietal, occipital, and temporal, that covers the outer layer of the brain (Hines, 2018). The frontal lobe is involved in executive function, ability to determine consequences from actions, and regulation of social responses by modifying emotions to fit socially acceptable norms (Hines, 2018). The prefrontal cortex, a region of the brain that is damaged frequently in TBIs, is located on the front part of the frontal lobe and is the main part of the brain responsible for an individual's will to live, cognitive behavior, decision making, social behavior, executive function, and personality expression (Hines, 2018). Executive function refers to the ability to differentiate between good and bad, conflicting thoughts, consequences to

potential actions, social "control", and etc…(Hines, 2018). Damage to the prefrontal cortex and subsequent symptomology will be studied in the following case studies (Hines, 2018). The temporal lobe of the brain, located inferior to the frontal and parietal lobe, is involved in processing sensory input for visual memory, emotion, and language comprehension (Hines, 2018). The limbic lobe, another important region of the brain impacted by TBI, is located on the medial surface of the cerebral cortex and consists of part of the frontal, parietal, and temporal lobes (Hines, 2018). The primary structures within the limbic system include the hippocampus, amygdala, thalamus, hypothalamus, basal ganglia, and cingulate gyrus, and are examples of deep structures that are connected to the cortex via pathways called white matter tracts (Hines, 2018). Hippocampus is involved in forming long-term memories and conflict avoidance, and the amygdala is involved in processing memory, emotion, and decision-making (Hines, 2018).

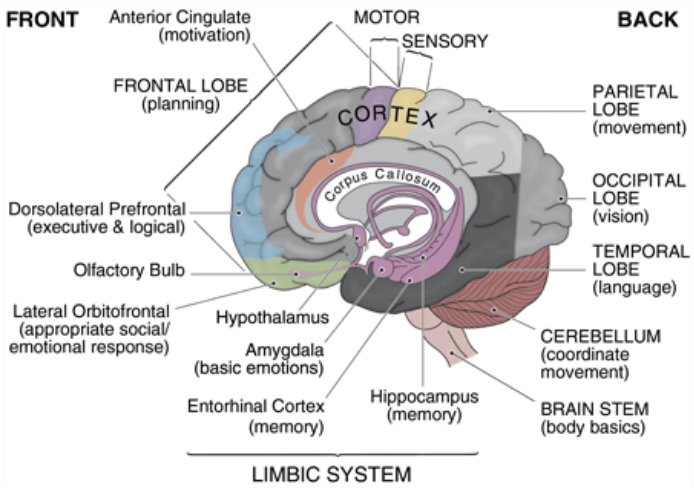

Diagram of the Brain

http://www.brainwaves.com/

The brain also has hollow fluid-filled cavities called ventricles that are filled with cerebrospinal fluid that flows within and around the brain and spinal cord as a shock absorber

from injury, a skull that protects the brain from injury, and is made up nerve cells and glial cells (Hines, 2018). Nerve cells convey information through electric and chemical signals via neurotransmitters, and glial cells provide the nerve cells with protection, structural support, and nourishments (Hines, 2018). Different types of glial cells include astroglia or astrocytes that regulate the blood brain barrier, oligodendroglia cells that produce myelin material that insulates axons to allow for faster transmittance of electric signals, ependymal cells that line ventricles and secrete cerebrospinal fluid, and lastly, microglia that function as immune cells and protect the brains against invaders (Hines, 2018).

Charles Whitman: Amygdala, aggression, and response to fear

a. Introduction

Charles Whitman, the UT shooter who climbed the UT tower and killed people with a sniper rifle, is a well-known example of a seemingly normal individual who committed a devastating crime (Wallenfeldt, 2018). He was an all-around smart, likeable guy before he started showing abnormal signs of rage and anger around the time he was married (Wallenfeldt, 2018). He started having spontaneous bursts of anger, once threatening to "kick the teeth out" of another person, was obsessed with note-taking, a condition associated with the amygdala called hypergraphia (Wallenfeldt, 2018). In these notes, he professed the overbearing love he had for his wife and scribbled down self-encouragement tips like, "CONTROL your anger. Don't let it prove you the fool. SMILE. Don't be belligerent. STOP cursing" (Wallenfeldt, 2018). The symptoms gradually became worse and oftentimes he would be overwhelmed with energy, almost manic, and be afflicted with horrible headaches (Wallenfeldt, 2018). His anger grew harder to control and he started having difficulties sleeping, concentration, and began to eat copiously (Wallenfeldt, 2018). He visited the UT Health Center for help but soon afterwards

gave into his compulsions and murdered his wife and mom before climbing the tower and killing 14 people (Wallenfeldt, 2018). He left several notes before he died, one of which being:

"I imagine it appears that I brutally killed both of my loved ones. I was only trying to do a quick thorough job...If my life insurance policy is valid please pay off my debts...donate the rest anonymously to a mental health foundation. Maybe research can prevent further tragedies of this type" (Wallenfeldt, 2018).

After his death, an autopsy revealed a glioblastoma tumor located beneath his thalamus, stretching from the hypothalamus into the temporal lobe, and impacting the amygdaloid nucleus (Wallenfeldt, 2018). However, Dr. Chenar, the doctor who had discovered the tumor, discovered the brain tumor was necrotizing and concluded that the tumor could not have significantly impacted Whitman's actions (Wallenfeldt, 2018). Dubious of this conclusion, the former Texas Governor John Connally sent a team of brain and mental health specialists to look further into Whitman's condition (Macleod). This investigation marked Dr. Chenar's report as incorrect and that the tumor could have contributed to Whitman's actions and emotions by putting pressure on the amygdala (Wallenfeldt, 2018).

b. Experiments linking Amygdala to fear response

The story of Charles Whitman wasn't the first to link damage to the amygdala and aggression. An experiment by John Downer in the late 1950s provided early evidence of the association between amygdala and violent behavior (Purves, Augustine, Fitzpatrick, 2001). Downer removed one amygdala from the brains of rhesus monkeys and disconnected the two hemispheres of the brain by transecting the optic chiasm, thus effectively removing all amygdala function from one half of the brain (Purves, Augustine, Fitzpatrick, 2001). He soon discovered that the monkey's behavioral response to the outside world became dependent on which eye they

used to view the world, one of which was linked to the part of the brain with amygdala function and the other lacking amygdala function (Purves, Augustine, Fitzpatrick, 2001). If the monkey was only using the eye not linked to the amygdala, they were placid when faced with humans (Purves, Augustine, Fitzpatrick, 2001). However, if the monkey was using the eye linked to the amygdala, they exhibited fearful and aggressive behavior (Purves, Augustine, Fitzpatrick, 2001). The monkeys reacted similarly if they were touched on either side of the body, which implies that the amygdala still had access to all sensory information (Purves, Augustine, Fitzpatrick, 2001). This experiment concluded that the amygdala "mediates processes that invest sensory experience with emotional significance" and evaluates stimuli, leading to more impulsive and angry behaviors (Purves, Augustine, Fitzpatrick, 2001).

Another experiment, conducted on rats, unveiled the role of the amygdala in evaluating stimuli (Purves, Augustine, Fitzpatrick, 2001). Joseph LeDoux and his colleagues at New York University conditioned fear responses in rats by pairing neutral stimuli, a sound, with an aversive stimuli, a foot shock (Purves, Augustine, Fitzpatrick, 2001). They would repeatedly play the sound and then administer a shock to the rat's foot immediately afterwards (Purves, Augustine, Fitzpatrick, 2001). After a period of time, the rats began to respond to the neutral stimuli, the onset of the sound, in the same adverse manner that they initially displayed towards the aversive stimuli, the foot shock (Purves, Augustine, Fitzpatrick, 2001). This adverse reaction towards the neutral stimuli was also accompanied with increased blood pressure and an increase in length of time animals crouch without moving, a behavior termed behavioral freezing, which occurs frequently in nature with prey animals as a last effort to escape predators (Purves, Augustine, Fitzpatrick, 2001). LeDoux traced the neuronal circuit that was utilized when the rats exhibited conditioned fear responses, and mapped out a pathway that went from the forebrain where the

auditory information was received to the medial geniculate nucleus of the thalamus and also between the medial geniculate and the amygdala (Purves, Augustine, Fitzpatrick, 2001). Furthermore, he realized if he destroyed the medial geniculate nucleus pathways, the fear response would still go through to the hypothalamus, given that the amygdala was still intact (Purves, Augustine, Fitzpatrick, 2001). His work confirmed that pathways leading from the amygdala to the reticular formation, a part of the brainstem that coordinates respiration and heart rate, was prominent in fear reactions, like the increase in blood pressure and freezing behavior seen in rats (Purves, Augustine, Fitzpatrick, 2001).

These studies show how the amygdala functions in learning about fearful stimuli. Without the amygdala, the rhesus monkeys were unable to properly differentiate between fearful and non fearful stimuli, and started to behave aggressively towards both types of stimuli. The pathway activated in the rat's brain during conditioned fear responses traveled through the amygdala, without which the rats were unable to exhibit signs of fear response, such as increased blood pressure and freezing behavior. The data from these experiments suggests a broader hypothesis that the primary function of the amygdala is to establish links between neutral stimulus and stimuli with primary reinforcement value to create an associative learning process, which is important in facilitating social behavior and making beneficial decisions in difficult circumstances (Purves, Augustine, Fitzpatrick, 2001). Indeed, another study discovered that subjects with damaged amygdala were unable to associate facial features, a neutral stimulus, with fear, an aversive stimuli, despite being able to associate all other types of facial expression accurately with different emotions (Purves, Augustine, Fitzpatrick, 2001).

Abnormal activation and electrical stimulation of the amygdala has been shown to result in fear/emotional reactions, sustained attention, and rage after termination of electrical

stimulation. Different case studies, dating back to the 1980s, have detailed individuals who developed aggression, rage, and fear after their amygdala was damaged. One such study details the journey of a man who developed anger management issues and pseudo-mystical and hyper-religious ideas after his amygdala was damaged following a head injury. However, the most significant reaction the amygdala controls is not hypothalamic activity but deeper neocortical processes that are responsible for the formation of emotional ideas. The amygdala can overtake the rest of the brain, including the neocortex, to form emotional ideas that the person will respond to with abnormal actions.

Aaron Hernandez: Chronic Traumatic Encephalopathy

a. Introduction

Aaron Hernandez, a former New England Patriots tight end, was an upcoming football superstar until he murdered a friend and two others in 2012 (Boston Globe). Previously, he had been one of the most dominant tight-end players, scoring at least five touchdowns each season for the Patriots (Boston Globe). His football career started in Bristol Central High School, where he made several state records playing as a wide receiver (Boston Globe). His history of head injury also started in high school, including one incident of a blindside hit so hard to the head that Hernandez was taken off the field (Boston Globe). After only three years of high school, he was drafted into the University of Florida football team and played there for three years, despite evidence of excessive marijuana and drug use. Although he was aesthetically prepared to play on a college football team, in no way was he academically and emotionally ready for college life. His coach and teammates found Hernandez to be “distressed” and “unhinged” and tried to push him in the right direction, but to no avail (Boston Globe). Other than his excessive drug use,

Hernandez was also struggling with his sexual identity and the death of his father, who abused him as a child (Boston Globe). Despite all of this, Hernandez excelled in football and was selected by the New England Patriots during the 2010 NFL draft (Boston Globe). During his NFL career, he received multiple injuries, including a hip injury, a sprained MCL, a knee injury, and multiple blows to the head. Outside of his career, he engaged in acts of aggression in nightclubs, took offense at the smallest things, and was paranoid that people were trying to fight him or physically challenge him (Boston Globe). In 2007, he was suspected of being involved in a shooting in Gainesville, and in 2012, of a double homicide in Boston (Boston Globe). He was indicted of murder charges of the killings but was later acquitted since there was no physical evidence tying Hernandez to the murders (Boston Globe). His long history of violence ended during the 2013 off-season when he was arrested and charged with the murder of his friend, Odin Lloyd, a semi-professional football player who had been dating Hernandez's fiancée's sister (Boston Globe). He continued to display aggressive and abnormal behavior in prison, screaming and banging on his cell door, and remained behind bars for four years, before he committed suicide (Boston Globe).

b. Chronic Traumatic Encephalopathy in the NFL

There have been multiple documented deaths of football players, including Andre Waters, Mike Webster, and Terry Long, attributed to Chronic Traumatic Encephalopathy, a neurodegenerative disease that was first introduced in an article in the *Journal of the American Medical Association* and described as a result of boxers "[taking] considerable head punishment seeking only to land a knockout blow" (Cantu, 2007). In other words, this disease occurs when there is significant and repetitive damage to the brain via concussions and blows, which tends to

happen in a lot of sports. According to a university study of retired athletes, sports players who had three or more concussions is three times more likely to experience memory problems, and those who had five or more is more likely to have earlier onset Alzheimer's disease (Cantu, 2007). The NFL had previously made the claim that there were no cases of CTE in the NFL in a Neurology journal, but this is no surprise as there were significant limitations to the study they conducted, including their biased sample population of young, active football players, difficulty in collecting data on LOC, variability in collection methods, and only information on concussions received during a small six year period during which the population of NFL players changed every year (Cantu, 2007).

c. Chronic Traumatic Encephalopathy: Pathology and Symptoms

After Hernandez's death, he was diagnosed with Chronic Traumatic Encephalopathy (CTE), a neurodegenerative disease that caused by repeated blows to the head. His brain had dark spots, withered and shrunken areas, significant damage to the frontal lobe, and indicated a severity of CTE that is the highest seen among cases with individuals his age (Boston Globe). Throughout most of his adult life, he displayed hallmarks of CTE, including poor judgment, lack of impulse control, paranoia, and aggression (Boston Globe).

CTE mostly results from sports playing, blast injuries during combat, and other types of neurotrauma, and is characterized by formation of clusters of neurofibrillary tangles from hyperphosphorylated tau protein, astrocytic tangles, and neurites around blood vessels in the cortex of the brain (McKee, Stein, Kiernan, Alvarez, 2015). However, it was only recently that the dangers of CTE became associated with popular, modern sports, such as football, ice hockey, boxing, and rugby, and the multiple concussions players often receive from these sports (McKee,

Stein, Kiernan, Alvarez, 2015). The phosphorylated tau protein changes indicative of early stage CTE were also found in those involved in combat who were exposed to blasts, injuries from which often include mild TBI, and multiple concussions, mirroring the early CTE pathology found in early adult football and soccer plays (McKee, Stein, Kiernan, Alvarez, 2015). The first people to be diagnosed with CTE was a group of 15 boxers who displayed enlarged ventricles, neurofibrillary tangles, neuronal loss, and cerebral atrophy (McKee, Stein, Kiernan, Alvarez, 2015). Corsellis, the one who first identified this pathology, summarized his findings by identifying areas of brain damaged and main symptoms, shown in Table 1.

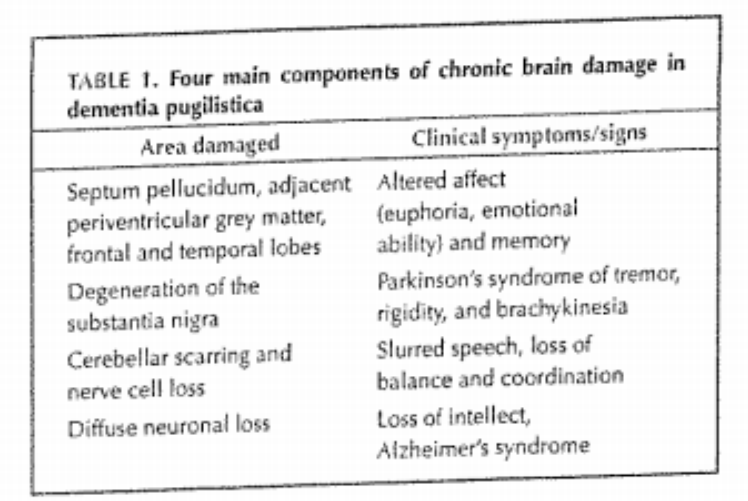

TABLE 1. Four main components of chronic brain damage in dementia pugilistica

Area damaged	Clinical symptoms/signs
Septum pellucidum, adjacent periventricular grey matter, frontal and temporal lobes	Altered affect (euphoria, emotional ability) and memory
Degeneration of the substantia nigra	Parkinson's syndrome of tremor, rigidity, and brachykinesia
Cerebellar scarring and nerve cell loss	Slurred speech, loss of balance and coordination
Diffuse neuronal loss	Loss of intellect, Alzheimer's syndrome

Table 1

Later, other cases of CTE documented predominance of neurofibrillary tangles in the hippocampus, brainstem, and other parts of the brain, abnormalities in phosphorylated TAR DNA-binding protein, and beta-amyloid plaques capable of strangling other neuronal cells (McKee, Stein, Kiernan, Alvarez, 2015). After many years and several more cases of CTE, a list of criteria for the diagnosis of CTE was developed (McKee, Stein, Kiernan, Alvarez, 2015). This list includes five hallmarks of CTE in the brain:

1. Phosphorylated tau protein immunoreactive neurofibrillary tangles surrounding blood vessels
2. Irregular distribution of phosphorylated tau proteins neurofibrillary tangles, astrocytic tangles, and dot- and thread-like neurites at the depths of cerebral sulci, consisting of grooves and furrows in the brain, and surrounding penetrating blood vessels
3. Neurofibrillary tangles in the crests of cerebral cortex located in layers II and III of the brain

As well as these two supportive features:

1. Clusters of astrocytic tangles in the cerebral cortex
2. Subependymal astrocytic tangles in the periventricular regions of the lateral ventricles, periaqueductal gray and lateral brainstem

The list of this criteria has been determined by a panel of certified neuropathologists as part of an initiative funded by the National Institute of Neurological Disorders and Stroke (NINDS) and the National Institution of Biomedical Imaging and Bioengineering (NIBIB). In early and mild CTE, the only noticeable changes of the brain are enlargement of the frontal and temporal horns of the lateral ventricles, interior cavities of the brain resembling a C-shaped structure , and increased perivascular, or surrounding blood vessels, in white matter (McKee, Stein, Kiernan, Alvarez, 2015). In intermediate and advanced CTE, identifiable changes include brain weight reduction, atrophy of gray and white matter, thalamus, hypothalamus, and mammillary bodies, a pair of small round bodies located on the undersurface of the brain functioning as part of the limbic system, enlargement of lateral and third ventricles, amongst others (McKee, Stein, Kiernan, Alvarez, 2015). There is very few macroscopic cerebellar abnormalities present in CTE as compared to the macroscopic pathology, including deposition of

phosphorylated tau protein in the form of neurofibrillary tangles in clusters around small blood vessels in the neocortex and as linear accumulations extending from the surface of the brain to deep layers of cortical gray matter (McKee, Stein, Kiernan, Alvarez, 2015).

A pathological progression of the criteria for CTE has been grouped into four stages (McKee, Stein, Kiernan, Alvarez, 2015). Those with Stage 1 CTE display post-concussive symptoms but is largely asymptomatic, and exhibits, though rarely, brain pathology of isolated perivascular epicenters of dot- and thread- like phosphorylated tau-protein neurofibrillary tangles and neuropil neurites (McKee, Stein, Kiernan, Alvarez, 2015). These tangles, if found, are located in the deeper parts of the cerebral grooves of different cortices, with a sparse quantity scattered throughout the adjacent cortex(McKee, Stein, Kiernan, Alvarez, 2015). Amyloid-beta plaques are not found in this stage in victims under the age of 50. Stage 2 CTE include subtle macroscopic changes, like slight enlargement of the frontal horns of the lateral ventricles and third ventricle, pallor of the locus coeruleus and substantia nigra, and the cavum septum pellucidum, in addition to more centers of tau-protein tangles (McKee, Stein, Kiernan, Alvarez, 2015). These tangles are also found scattered throughout the superficial cortical layers in the adjacent cortex (McKee, Stein, Kiernan, Alvarez, 2015). Beta-amyloid plaques are found in 19% of those older than 50 years old, and clusters of reactive microglia, axonal swellings, and distorted subcortical U-fibers are also pathological signs in this stage (McKee, Stein, Kiernan, Alvarez, 2015). Brains with stage 3 CTE show more macroscopic changes, including reduction in brain weight, atrophy of frontal and temporal lobes, and enlargement of lateral and third ventricles. 50% of the affected have septal abnormalities, atrophy of various structures, and patches of tangles are visible around blood vessels at sulcal depths in multiple cortices (McKee, Stein, Kiernan, Alvarez, 2015). Lastly, brains in the most severe stage, stage 4, show a

substantial decrease in brain weight, cerebral atrophy, severe thinning of hypothalamus floor, atrophy of various structures, septal abnormalities, widespread myelin and neuronal loss, etc… Tau protein tangles are clustered and densely distributed through multiple parts of the brain.

Phineas Gage: Psychopathy with Brain Injury

The curious tale of Phineas Gage provides the earliest evidence of the role of the prefrontal cortex in personality alteration. Phineas Gage's medial prefrontal cortex was injured when a tamping iron blast through the left side of his face during a worksite accident (Koenig, 2014). Within the next four months, he nearly recovered full physicality, but his personality was drastically altered (Koenig, 2014). He changed from an energetic, likeable man to someone who was fitful, tactless, and profane (Koenig, 2014). In fact, his personality became so different that his friends said he was "no longer Gage" (Koenig, 2014). There have been some exaggerations of the extent of his personality change, but his brain injury did alter, to some extent, who he was as a person (Koenig, 2014).

Since Gage's time, it has been scientifically determined that the ventromedial prefrontal cortex and anterior cingulate cortex have roles in inducing psychopathy, which might have been the case for Phineas Gage (Koenig, 2014). Psychopathy is a personality disorder in which the affected show symptoms of impulsive antisocial behavior and lack of empathy (Koenig, 2014). Not only do criminals with psychopathy show less remorse for their crime and are more violent, they are more likely to commit other crimes after rehabilitation (Koenig, 2014). The prevalence of psychopathy is 1% in the general population, but 15-25% in the prison population, meaning that psychopaths tend to commit a disproportionate amount of crime (Koenig, 2014). A better

understanding of the psychobiological mechanisms involved with this disorder will lead to more effective treatment plans for those who are psychopathically influenced.

The first person who described this personality disorder was Hervey Cleckley, an American psychiatrist who outlined hallmark characteristics of individuals in his psychiatric practice who had no cognitive defect or mental illness but had a severe disregard for the rights and welfare of others (Koenig, 2014). Cleckley said, "My concept of the psychopath's functioning postulates a selective defect or elimination which prevents important components of normal experience from being integrated into the whole human reaction, particularly an elimination or attenuation of those strong affective components that ordinarily arise in major personal and social issues" (Koenig, 2014). Some of the characteristics he pointed out included: Untruthfulness and insincerity, lack of remorse or shame, failure to learn by experience, poor judgement, and unresponsiveness in interpersonal relations, traits that are similar to those experienced by victims post-TBI (Koenig, 2014). In other words, TBI might induce psychopathic traits in certain individuals, like Phineas Gage, which could lead them to become future offenders (Koenig, 2014).

a. The Ventromedial Prefrontal Cortex

The ventromedial prefrontal cortex (vmPFC), a specific part of the prefrontal cortex that is located at the at the bottom of the cerebral hemisphere in the frontal lobe, we now know processes risk, fear, decision-making, and self-control, although there is still no clear consensus on it's main function (Koenig, 2014).

In 1975, scientists discovered that injury to this specific part of the prefrontal cortex resulted in "pseudopsychopathy" in many neurological patients (Koenig, 2014). Antonio

Damasio, a Portuguese-American neuroscientist, researched this link between vmPFC injury and pseudopsychopathic changes (Koenig, 2014). He discovered that the ventromedial prefrontal cortex (vmPFC) was responsible for emotional and decision-making capabilities, and that damage to this area would result in lessened degrees of shame, guilt, and empathy, as well as poor planning skills, irresponsibility, and failure to learn from mistakes (Koenig, 2014). Individuals with lesions to the vmPFC area and psychopaths both display reduced reactions to emotionally charged stimuli and reversal learning deficits, and changes in decision making (Koenig, 2014). His first hypothesis regarding the functions of the vmPFC was termed the somatic marker hypothesis, which states that the vmPFC marks decision outcomes as something to pursue or avoid based on its associations with somatic markers, including emotional associations and associations between mental objects and bodily feedbacks, to use in decision-making (Koenig, 2014). This hypothesis also lends vmPFC a role in regulating emotions and emotional reactions, since the vmPFC would be the part of the brain to label each somatic marker as positive and negative, which affects the individual's emotional response to each marker (Koenig, 2014). Damasio concluded that the vmPFC regulates certain aspects of psychopathy and low-anxious psychopaths are comparable to those with damaged vmPFC (Koenig, 2014).

The somatic marker hypothesis, supported by neuropsychological and electrodermal data, is just one of the theories on vmPFC function (Koenig, 2014). A second hypothesis, supported by recent functional neuroimaging data, states that the function of vmPFC is to quantify the value of goal-directed outcomes and options (Koenig, 2014). A third hypothesis, created through analysis of human imaging data and data from fear conditioning paradigms, states that vmPFC regulates negative affect through top-down inhibition, such as fear and anxiety, by inhibiting the

amygdala, a portion of the brain that processes emotional and social information (Koenig, 2014). This hypothesis was created using human imaging data and data from fear conditioning paradigms (Koenig, 2014). However, this hypothesis is in direct contradiction to the present "low fear hypothesis", or the belief that vmPFC dysfunction lowers the degree of fear and would lead to the affected developing psychopathic qualities (Koenig, 2014). In addition, according to the third hypothesis, a damaged vmPFC would not lead to psychopathy (Koenig, 2014). Lastly, a fourth model states that vmPFC is the basis of self-reflection and rumination and is responsible for manifesting guilt, embarrassment, and empathy (Koenig, 2014).

b. The Anterior Cingulate Cortex

Another region of the prefrontal cortex that plays a role in psychopathy, the anterior cingulate cortex, oversees behavioral control and and regulates behavior through cognitive and affective mechanisms (Koenig, 2014). It has connections to the limbic system, a region of the brain involved in emotion processing, other regions of the prefrontal cortex, such as the orbitofrontal cortex that has reward-related functions, autonomic systems, such as the hypothalamus and brainstem, and the hippocampal region responsible for memory (Koenig, 2014). Therefore, it is essential in regulating affect and holds many functions (Koenig, 2014). Activity in this brain region is associated with empathy, error detection, pain, reward, emotion, and social behaviors (Koenig, 2014). Those with damage to the ACC, such as cingulotomy patients, exhibit reduced motivation and response to emotional stimuli in addition to greater social disinhibition and irritability (Koenig, 2014).

c. Functional Neuroimaging in the Prefrontal Cortex

Neurological imaging provides evidence linking alterations in the vmPFC and ACC with psychopathic behavior. Present day MRI data can yield measures of the volume, thickness, and density/concentration of gray matter (Koenig, 2014). Different studies comparing psychopathic and non-psychopathic control groups have established an association between psychopathy and reductions in PFC gray matter. In one such study, grey matter volume was compared between three groups: "unsuccessful" psychopaths, "successful" psychopaths, and a non-psychopathic control group (Yang, et al., 2010). The data established a negative correlation between gray matter volume and factor of psychopathy, with the unsuccessful psychopaths having much lower yields of volume compared to successful psychopaths and non-psychopaths (Yang, et al., 2010). In another study conducted by the same group of researchers, they discovered that gray matter was reduced especially in the lateral PFC, ventral PFC, and orbitofrontal cortex in unsuccessful psychopaths, but not reduced at all in the successful psychopath group when compared to the control group (Yang, et al., 2010). In a third study, they discovered that cortical thickness was also negatively correlated with increased facets of psychopathy (Yang, et al., 2010).

One type of functional imaging study, called resting-state functional connectivity MRI, found reduced functional connectivity between the dorsal ACC and insula as well as the amygdala and anterior vmPFC in psychopaths by measuring activity between brain regions (Koenig, 2014). Numerous other fMRI studies, involving multiple stimuli and response paradigms, also link psychopathy with activity in the vmPFC and ACC during social-affective processing (Koenig, 2014). The studies also show how individuals might be biologically predisposed to display more symptoms of psychopathy than others based on the contents of their brain or how thick it is, and how the prefrontal cortex is a definite substrate for psychopathic disorder. A more comprehensive understanding of how this important region of the brain links to

psychopathy will help offenders displaying this disorder, and, as stated by Cleckley, "the cause of the psychopath's disorder has yet been discovered and demonstrated. Until we have more and better evidence than is present available, let us admit the incompleteness of our knowledge and modestly pursue our inquiry" (Koenig, 2014).

Ricky Green: Psychopathy without Brain Injury

a. Introduction

Ricky Green is a good example of an individual who was psychopathic without sustaining any sort of brain injury. Hare, the author of *Bad without Conscience,* states that psychopaths "lack a conscience, the ability or sensitivity to shape a moral issue and to understand the significance of such issues, and to experience empathy," are prone to deception and violence, and can commit acts of extreme immorality. By the time Green turned 29, he had sexually mutated and murdered two women and two men without any reason or cause (Hare, 1999).

Ricky Green was born in Fort Worth, Texas, into a dysfunctional childhood replete with sexual and physical abuse and violent behavior (Hare, 1999). He had an intense fear of his father, who was harsh and demanding and beat his younger sister and mother, and participated in abnormal activities, such as killing animals, setting things on fire, indicative of antisocial and psychopathic tendencies (Hare, 1999). Green started drinking and using drugs from a very early age and was sexually abused by an older man who kidnapped him and his grandfather (Hare, 1999). His later sexual experiences, with mostly women, would exacerbate his psychopathic behavior and disinhibit his murderous impulses (Hare, 1999).

He met his first victim, Jeffrey Davis, after Davis had run away from home to be with his girlfriend (Hare, 1999). A month after the initial meeting, they met again at Davis' house, where Green went "berserk" after Davis made a sexual advance at him. Unable to stop himself, Green stabbed Davis multiple times, sexually mutilated him, and tried to behead him. Immediately afterwards, Green felt the weight of what he had done and confessed to his wife. His second victim, Steve Fefferman, was murdered after he invited Green to his house, performed oral sex on him, and confessed to having pedophilic tendencies. Fefferman's sexual advances and stories repulsed Davis and led Davis to viciously murdering and sexually mutilating him later that night. After committing the murder, Davis once again called his wife to confess and felt remorse for what he had done. However, this remorse didn't stop him from locating his next two victims, Betty Joe Monroe and Sandra Bailey, who he murdered on two separate occasions after engaging them in sexual activity. He did not feel as guilty about his latter crimes than he did with his formers one and asked his wife to clean up after his last two murders instead of apologizing.

Green was charged with these murders, sentenced to death by the Texas trial court, and subsequently executed. During his execution, Green stated that he felt like he was no longer a "threat to society," and thanked the "Lord for giving me the opportunity to get to know Him" (Hare, 1999).

b. Culpability

It is unclear whether people are biologically predetermined to have psychopathic tendencies or are bred to become psychopaths. Some believe that psychopaths have free will and have made decisions that resulted them into becoming who they are. Others believe psychopaths have different brain structures that predispose them into acting the way they do. Either way, it is

difficult to determine the extent of free will Ricky Greene had over his actions. Green had described himself, while committing these murders, as being in an "altered" state. He not only killed his victims but did so maliciously, stabbing and mutilating the body after dealing the killing blow. (Hare, 1999). However, there have been incidents of Green tending to kill a sexual partner but successfully avoiding it at the last minute, once due to a sudden rush of self-awareness and another time after his target spoke of his son, who was coming home soon (Hare, 1999). In each of these incidences, something had caused Green to snap out of his "altered" state and consider the moral implications of his actions. It is possible that Green's conscious decisions to increase drug use and drink heavily later suppressed his morality and rendered him unable to snap out of his murderous intent when committing his murders. In fact, Green had admitted in prison that alcohol "allowed him" to kill others, and indeed, his murders were all committed under the influence of alcohol (Hare, 1999). Many factors could have exacerbated Green's murderous urges, including the physical and sexual abuse he endured in his childhood, his hatred towards prostitute and homosexuals, and the crime-ridden environment he was raised in. However, given the number of individuals with unfortunate childhoods who did not commit murder later in life, these factors cannot excuse Green for the murders he committed. Green was aware of the crimes he was committing but was unable to control himself and made no effort towards self-intervention after his first couple murders. The case of Ricky Green illustrates an individual, though aided by unfortunate scenarios and is mentally disordered, who is fully criminally responsible for his actions.

PART THREE: TBI IN THE CRIMINAL JUSTICE SYSTEM

Prevalence of TBI in Prison Populations

TBI rates have been proven to be more elevated in inmate populations, ranging from 25-87%, as compared to much lower 8.7% found in the general population, meaning that many inmates have cognitive, behavioral, and emotional difficulties (Williams, Mewse, Tonks, Mills, Burgess, & Cordan). The results of a study designed to determine rates of TBI in a random sample of adult offenders indicated that approximately 65% of the inmates had suffered a TBI, 16% of whom experienced moderate and severe TBI (Williams, Mewse, Tonks, Mills, Burgess, & Cordan). Different research studies have affirmed TBI as a potential causal factor for likelihood of initial offending, which is especially clear given the high frequency of individuals who are or were incarcerated and had experienced TBI.

One case involving juvenile subjects, conducted by Mckinlay in 2013, showed how childhood TBI is predictive of future offending behavior (McKinlay, Albicini). He organized the subjects into three groups: Moderate/Severe TBI group, Mild TBI group, and a control group consisting of children who had orthopedic fractures but not brain injuries (McKinlay, Albicini). All these injuries had occurred five years prior to the study so that ensuing offending behavior could be measured (McKinlay, Albicini). Among the three groups, the Moderate/Severe TBI group was more likely to have a history of offending behavior, arrest, conviction, and petty crime (McKinlay, Albicini). He also conducted another study that looked at the association between individuals who had a TBI during their childhood and adult offending behavior (Brain Injury Association). The results from this study revealed a positive correlation between TBI and future offending behavior and discovered that adults who experienced childhood TBI were more likely

to commit offensive behavior as an adult (Brain Injury Association). Another study conducted by Perron and Howard found that delinquents with TBI tend to exhibit more psychiatric distress, earlier onset of criminal behavior, earlier onset of substance abuse behavior, and more lifetime substance abuse and suicidality (Brain Injury Association).

Although these aforementioned studies may not establish a definite causality between TBI and violence, it definitely makes theoretical sense in how some of the downstream consequences of TBI, such as behavioral problems, could predispose victims to future offending behavior. In addition, individuals post-TBI experience similar issues that incarcerated individuals experience, such as reduced coping skills, impulse control, emotion regulation, etc...Thus, there is a good reason to believe that this relationship exists and that this problem is incredibly prevalent in the prison population. The findings from these studies makes it clear that there is a need to account for TBI in the assessment and management of offenders (Williams, Mewse, Tonks, Mills, Burgess, & Cordan).

Impact of TBI in Prison Populations

The impact of TBI in a prison setting has not yet been carefully analyzed but can potentially be great (Wald, Helgeson, & Langlois, 2018). TBI is already overrepresented in prison populations, and inmates with no history of TBI are also at risk of receiving TBI while in prison or jail. For example, in the Minnesota project, incarcerated gang members were reported to have their heads beaten as part of an initiation process (Wald, Helgeson, & Langlois, 2018). In another case, inmates were reported to have knocked their heads against the bars until they became unconscious (Wald, Helgeson, & Langlois, 2018).

There are many short- and long-term effects of TBI, including a wide range of problems in thinking, sensation, language, emotions, learning...TBI often results in cognitive, social, emotional, and behavioral problems (Wald, Helgeson, & Langlois, 2018). It is not surprising that the deficits associated with TBI has a huge, and mostly negative, impact on behavior in prison. Attention deficits are very common, especially among adolescents, and manifests itself in

difficulty focusing on or responding to required tasks or directions (CDC). Memory deficits make it difficult to remember rules or directions given by a correction officer (CDC). Irritability and aggressiveness can lead to violent confrontations (CDC). Slowed verbal and physical responses can be perceived as uncooperation and disrespect (CDC). Uninhibited or impulsive behavior include problems controlling anger or sexual urges and can lead to further altercations (CDC). All of these disciplinary problems make it difficult for detained inmates with TBI to make a smooth transition to prison life and rules, and many officials may also attribute these behavioral problems to mental illness (CDC). In addition, aggression and violent behavior also leads to increased risk for recidivism, meaning that inmates who are aggressive as a result of TBI are more likely to commit crimes after release (Wald, Helgeson, & Langlois, 2018). This is another reason why effective screening for TBI-related behavior problems is necessary so inmates with a history of TBI can be properly managed and treated (Wald, Helgeson, & Langlois, 2018).

Although not all TBI result in long-term disability, there are many TBI-related secondary conditions, such as substance and alcohol abuse, which could lead to problems during re-assimilation into society (Wald, Helgeson, & Langlois, 2018). It has been proven that individuals with history of TBI are much more likely to have problems with drugs and alcohol (Wald, Helgeson, & Langlois, 2018). However, due to lack of identification of TBI, many also lack the knowledge that these problems could stem from TBI. Increased awareness of TBI and associated problems could help facilitation implementation of TBI-specific interventions, resulting in formation of secondary programs for rehabilitation to help inmates reintegrate successfully into society (Wald, Helgeson, & Langlois, 2018).

Changes to the Criminal Justice System

Despite the high rate of TBI in inmate populations and positive correlation between TBI and violence, there is still a lack of validated, standard, screening procedure, psychoeducation for corrections officials, and lack of medical attention. In one study conducted by Pamela Diamond,

she discovered that initial screening methods only detected TBI in 19% of the inmates that were a part of her sample population during the Minnesota study (Wald, Helgeson, & Langlois, 2018). Only 10 out of the 998 inmates who all reported a history of TBI screening had reported it during their intake screening (Wald, Helgeson, & Langlois, 2018). 816 more inmates reported incidence of at least one brain injury while taking the TBIQ, a questionnaire geared towards TBI identification (Wald, Helgeson, & Langlois, 2018). This goes to show how a more detailed and thorough screening is imperative in accurately identifying inmates with a history of TBI.

A validated screening tool is necessary to ensure that results are free of false negative and positives, such as The Traumatic Brain Injury Questionnaire (TBIQ) and The Ohio State University TBI Identification Method (Wald, Helgeson, & Langlois, 2018). The former consists of a one-to-one interview with three different sections of questions (Wald, Helgeson, & Langlois, 2018). Section 1 includes questions on the presence of previous head injury experiences resulting from 12 different situations (vehicle crashes, assaults, etc…) (Wald, Helgeson, & Langlois, 2018). Section 2 goes more into depth on the details of the head injuries and inquires about the year of the injury, whether or not there was a loss of consciousness, and what care was received (Wald, Helgeson, & Langlois, 2018). Section 3 gauges the the severity of 15 common physical and cognitive symptoms resulting from head injury, like amnesia, vertigo, headaches, etc... (Wald, Helgeson, & Langlois, 2018). This screening instrument takes around 15 minutes to administer and is validated correctional populations (Wald, Helgeson, & Langlois, 2018). However, it may take much longer if the user has had several TBI in the past (Wald, Helgeson, & Langlois, 2018). The Ohio State University TBI Identification Method is similar but consists of only two steps (Wald, Helgeson, & Langlois, 2018). During Step 1, the interviewee is asked to recall head injuries resulting from blows to the head or neck from assailants or high-velocity forces (Wald, Helgeson, & Langlois, 2018). During Step 2, the interviewee is asked about details regarding each incident and questions similar to those asked in Section 2 of the TBIQ (Wald, Helgeson, & Langlois, 2018). Even though the OSU is less extensive, it takes only about 5 minutes to administer (Wald, Helgeson, & Langlois, 2018). Another test, the HELPS TBI, also

takes about 5 minutes to administer (Wald, Helgeson, & Langlois, 2018). Different screening methods are useful in different correctional settings and depends on the volume of intake screenings (Wald, Helgeson, & Langlois, 2018). For example, the shorter OSU screening instrument would be more useful in efficiently analyzing a high intake population (Wald, Helgeson, & Langlois, 2018).

One of the main reasons TBI assessment has not yet been incorporated into initial screening procedures is the sheer amount of inmates admitted into jails and prisons every year. In addition, time and effort is required to give each inmate a full history and physical, which is more comprehensive and reveals more data than a TBI assessment, so measures have not been taken to appropriately identify inmates with TBI (Wald, Helgeson, & Langlois, 2018). In addition, the TBI screening measures discussed above will only identify presence of TBI and are not designed to determine whether specific deficits in function are present (Wald, Helgeson, & Langlois, 2018). Additional, more advanced, screening procedures are necessary to determine severity of TBI and small subgroups of TBI victims, so subsequent problems, such as specific loss of function or impaired mental state, can be linked back to TBI (Wald, Helgeson, & Langlois, 2018). One such screening measure, the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), can assess for presence of cognitive deficits resulting from TBI (Wald, Helgeson, & Langlois, 2018). Other assessments, like evaluations designed to identify TBI-related secondary problems, are also necessary in identification of appropriate treatment for co-occurring conditions (Wald, Helgeson, & Langlois, 2018). Incorporation of a general TBI screen is already difficult but addition of subsequent, more complex, screening measures will take up even more resources (Wald, Helgeson, & Langlois, 2018).

Despite these obstacles, proper screening techniques can be incorporated. The “TBI in MN Correctional Facilities: Strategies for Successful Return to Community” is an example of a successful TBI identification pilot project that managed to enhance facility and community safety through identification and intervention for offenders with TBI (Wald, Helgeson, &

Langlois, 2018). This project shows how increased cooperation between TBI and criminal justice professionals could increase identification, rehabilitation, and treatment of inmates with TBI.

In addition to implementation of proper screening measures, it is also important to educate correction officials on the significance of TBI in their inmates populations by providing them with resources, such as a CDC fact sheet aimed at educating criminal justice professionals, so they can understand how to better manage inmates with TBI and recognize the consequences of TBI, such as memory deficits and slowed verbal responses, which they could otherwise falsely interpret as uncooperation and disrespect.

Lastly, improving treatment and management of rehabilitation programs and solutions is a necessary second step after identification and education (Wortzel, Arciniegas, 2012). Secondary programs that target relieving secondary symptoms, such as substance abuse, depression, and homelessness, of TBI should be formally established to lead to a higher success of treatment (Wortzel, Arciniegas, 2012). Programs that target specifically victims of TBI should also be established. An ideal Brain Injury Rehabilitation program would involve a multidisciplinary team providing services like physical therapy, occupational therapy, speech therapy/cognitive retraining, neuropsychology, neuropsychiatry, social work, recreational therapy, physiatry, neurology (Wortzel, Arciniegas, 2012). Physical therapy involves anything located on or below the waist, and works at building mobility, endurance, and balance (Wortzel, Arciniegas, 2012). Occupational therapy pertains to anything from the waist up and helps people adjust back to their regular activities, such as therapy targeted towards improving visual perception skills to help victims start driving again (Wortzel, Arciniegas, 2012). Speech therapy focuses on memory retainment, language, concentration, and swallowing (Wortzel, Arciniegas, 2012). Additional therapy should help resolve ailments such as dizziness in the vestibular, or inner ear, system, a common symptom amongst victims with mild TBI (Wortzel, Arciniegas, 2012). These services would be extremely helpful for inmates with TBI during the first 4-6 months (Wortzel, Arciniegas, 2012). There are also other types of services, such as pharmacotherapies and medications, that target resolving cognitive impairments, loss of memory

and processing speed, etc...that can be expanded on and be made more accessible as well (Wortzel, Arciniegas, 2012).

PART FOUR: APPLICATION TO INDICTMENT IN THE CRIMINAL JUSTICE SYSTEM

The Legal Concept of Responsibility

The end goal of this thesis is to determine the conditions that stipulate whether a person should be held criminally responsible for their actions. Legally, a defendant is criminally responsible for his crime only if he committed the crime, also known as *actus reus*, with a guilty mind, also known as *mens rea*, without affirmative defenses, which includes the insanity defense, defense of others, duress, etc…For example, second-degree manslaughter is charged when someone causes the death of other, which constitutes a guilty act, but recklessly, which does not constitute a guilty mind as the death was not premeditated or intended upon (New York Penal Code). In contrast, second-degree murder is charged when someone causes the death of other, a guilty act, with the intent of killing them, a guilty mind (New York Penal Code). The latter charge would result in harsher consequences than the former. That means that not only should courts consider the actions of the offender, but also if his mind was engaged in and had full control of his actions. As mentioned before, if the criminal qualifies for the insanity defense, they will not be held criminally responsible and cannot be subject to punishment other than commitment to a mental institution and treatment. However, there are many cases of individuals who lurk in the gray zone. Individuals who were cognizant of their criminal actions but also were not in full control over them. New neuroscientific results, such as MRIs, can help identify this class of people who do not qualify for the insanity defense but also fail to meet a condition of criminal responsibility. People with altered brain function due to traumatic brain injury would fall into this category. These results could help the mentally ill receive lesser charges and less severe punishments. Further research on neuroscientific studies would help better these standards and shed light on the neural mechanisms responsible for criminal behavior.

a. Voluntary Action

A voluntary action, the *actus reus* of crimes, must be guided by a conscious mental thought (Maoz & Yaffe, 2016). For example, if someone were to accidentally injure another while flailing around during an epileptic seizure, they should not be held responsible for the injury since they did not mentally instruct their body to move the way it did (Maoz & Yaffe, 2016).

Several experiments have been done in the hopes of knowing if bodily movements are guided by conscious brain activity (Maoz & Yaffe, 2016). In one experiment, conducted by Benjamin Libet, individuals' brain activity were monitored by an electroencephalography when asked to move their hand (Maoz & Yaffe, 2016). They were placed in front of a rapidly moving spot on a clock and were asked to recollect the position of the spot when they first felt the urge to raise their hand (Maoz & Yaffe, 2016). According to this data, this urge occurred about 200 milliseconds before they made any movement (Maoz & Yaffe, 2016). However, the readiness potential, a electric potential in the cerebral cortex, activates around 500ms before onset (Maoz & Yaffe, 2016). This shows that brain activity preludes conscious thought before performing an action, and that unconscious brain activity might initiate movement (Maoz & Yaffe, 2016). This also shows that individuals might be tricked by their brain into thinking that they're acting of their own free will when they're not (Maoz & Yaffe, 2016).

Another experiment tested individual's degree of "sense of agency", or awareness they have when initiating motor movement, in relation to the behavior it accompanies (Maoz & Yaffe, 2016). The subjects were patients who had undergone brain surgery and needed invasive brain mapping to minimize complications from surgery (Maoz & Yaffe, 2016). During the brain mapping process, the subjects would have different regions of their brain stimulated electrically and, in some cases, individuals would have the urge to perform an action after stimulation (Maoz & Yaffe, 2016). It was discovered that right inferior-parietal stimulation provokes a desire for contralateral movement while left inferior-parietal stimulation triggers lip movement and speech (Maoz & Yaffe, 2016). After stimulation, some of these patients thought they performed the

movements when they had not (Maoz & Yaffe, 2016). This is evidence that an individual's "sense of agency" may not directly translate into a voluntary action, meaning that an individual's willingness to do something may be an unreliable guide to the voluntariness of the behavior that follows. In addition, stimulation of the premotor-region induced slight contralateral limb and mouth movements without the subjects knowing (Maoz & Yaffe, 2016). This experiment shows that the brain circuitry connected with movement is not closely related to the one connected with sensation (Maoz & Yaffe, 2016). In other words, it is possible to experience agency over phantom actions and perform actions with no sense of agency (Maoz & Yaffe, 2016). Individuals with this problem would not qualify under the law's voluntary act requirement, since their motor movements would not be preceded by conscious mental activity. Even though this by no means disqualifies the law's definition of voluntary act, it is something noteworthy that could be deemed applicable to some offenders in the future and help them receive lesser charges.

b. Intention and the perception of risk

Imagine that someone sells Joe a flat screen TV for an extremely cheap price of 200 dollars. Joe takes the opportunity and makes the exchange but later is arrested for possession of stolen goods. In this situation, would Joe be criminally responsible for theft? This depends on the *mens rea* standard upheld in court (Maoz & Yaffe, 2016). There are numerous ways to perceive a given fact, like the fact that a property was stolen. Is Joe guilty only if he's fully aware of the guilt behind the action or would he still be convicted if he had some inkling of an idea? The two most relevant types of *mens rea*, employed in the Model Penal Code, includes intent and recklessness. Intent is the mental act of consciously aiming at an action to achieve a goal (Maoz & Yaffe, 2016). Recklessness is "awareness of a substantial and unjustifiable risk" as a consequence of an action (Model Penal Code).

Experiments have led to the discovery that accumulation of firing rate of neurons, located in the middle temporal areas of the lateral intraparietal area, towards a threshold increases the probability that there is an aim at a finalized perceptual judgment (Maoz & Yaffe, 2016). In one

such experiment, scientists had monkeys determine the overall direction of many dots moving in different directions. As the monkeys got closer to reaching their decision, their neuronal firing rates build up towards a threshold reflecting on accumulation of evidence towards a finalized decision, or perceptual judgment (Maoz & Yaffe, 2016). However, a perceptual judgment is different from intention, given how the monkey didn't need intent to reach their decision. Although, if the monkeys had received an award after reaching their decision, their decision might involve intent as well.

Another study that better measures intent had it's subjects decide whether they preferred to add or subtract two unknown double-digit numbers (Maoz & Yaffe, 2016). After the subjects mentally decided to add or subtract, they were presented with the numbers for two seconds, then a screen that contained the sum and difference of the numbers and some distractor numbers (Maoz & Yaffe, 2016). The subjects would then choose a number, based on their initial decision to add or subtract the two numbers (Maoz & Yaffe, 2016). The experimenters were able to determine each subject's initial decision with 71% accuracy when decoding from the anterior medial prefrontal cortex and 60% accuracy when decoding from the lateral prefrontal cortex (Maoz & Yaffe, 2016). However, it is still not certain whether intent is actually coded in these regions, as activity in these regions might just accompany the subject's intent to add or subtract (Maoz & Yaffe, 2016). Intention has not yet been a mappable as a single neural process, and further work is needed to determine a neural process that can distinguish intention from other mental states that accompanies sensory information (Maoz & Yaffe, 2016).

c. Self-Control

The justice system also accounts for the level of self-control, or free will, that one possesses when committing a crime (Maoz & Yaffe, 2016). Typically, criminal law is insensitive to whether one could have been in control if they stopped to think at an earlier time versus one who just lost control entirely (Maoz & Yaffe, 2016). However, these two scenarios do differ from a moral point of view. Usually the law grants mitigation in homicide cases with heightened

emotion rather than destruction of property with heightened emotions (Maoz & Yaffe, 2016). Under one formulation of the insanity defense, individuals who find it extremely difficult to do what the law requires them to do is excused from criminal punishment (Maoz & Yaffe, 2016). The Supreme Court has also ruled that adolescents who have committed serious crimes cannot be punished to the same extent as adults as they have less self-control (Maoz & Yaffe, 2016).

There are many discrepancies in the ruling of offenders when it comes to self-control. The Court is more lenient on individuals with no self-control when it comes to homicide cases, which there are far fewer of, and not cases involving destruction of property (Maoz & Yaffe, 2016). This conveys the rather stingy attitude of the Court when it comes towards basing differences in treatment on differences in control (Maoz & Yaffe, 2016). Courts also seem to predicate a different in treatment of adolescents, though not adults, on a difference in the capacity of self-control (Maoz & Yaffe, 2016). More neuroscientific work needs to be conducted on the factors that influence self-control, so the legal system can predicate more differences in treatment under the law on differences in control.

Criminal Justice System Procedure

Both criminal law and criminal procedure dictate the consequences and agenda of criminal cases, though the outcome of a case is mostly dependent on the criminal procedure and the details of the case as opposed to the actual substantive law, which varies greatly by state. Therefore, a basic understanding of criminal procedures, terminology, and game changing areas of potential impact at different stages of the criminal process is helpful before further understanding the relationship between TBI and culpability.

Offenders with altered brain function, including TBI and mental illness, are usually arrested or incarcerated on two types of criminal violations, misdemeanor and felony charges, or because a family member had called the police due to the offender performing dangerous or threatening actions (National Alliance on Mental Illness, 2017). There is no universal definition to categorize misdemeanors and felonies, as it is usually decided by the police who first arrived

on the scene based on the evidence and extent of harm to others, but misdemeanors are generalized as crimes that are punishable by imprisonment of one year or less while felonies are crimes that are punishable by imprisonment of more than one year (National Alliance on Mental Illness, 2017).

Let us discuss the proceedings of the criminal justice system with a fictional offender, Joe, who has had a traumatic brain injury in the past. After Joe commits a crime, the police that arrive at the scene has several options to choose from. They can let Joe go with a warning if there was no physical injury and not take further action (National Alliance on Mental Illness, 2017). They can also take Joe into custody and have him examined by mental health authorities with possible commitment to an institution (National Alliance on Mental Illness, 2017). This option does not constitute an arrest, though it will end with confinement into a psychiatric facility for one to three days before being released back to the community with or without future plans of treatment (National Alliance on Mental Illness, 2017). The police can also issue the offender a citation to appear in court later without arresting him (National Alliance on Mental Illness, 2017). The three options above can only be considered if the police deem the crime a misdemeanor and not a felony, which entails more serious consequences (National Alliance on Mental Illness, 2017). The last option they have is to arrest the offender and transport him to the police station, which can occur in the case of both a misdemeanor and a felony, if they find the offender's actions disturbing or evidence of physical injury at the scene (National Alliance on Mental Illness, 2017).

In the last option, an arrest only officially occurs if and only if Joe is put under custody by the police to be charged with the crime (National Alliance on Mental Illness, 2017). In other words, if the police takes Joe into custody without the intent of charging him with a crime, he is not arrested. The police can only make a lawful arrest, supported by probable cause, if they believe that a crime has been committed and committed by Joe (National Alliance on Mental Illness, 2017). In the case of a felony charge, the police can use evidence from the crime scene and witnesses to make a lawful arrest (National Alliance on Mental Illness, 2017). However, in

the case of a misdemeanor charge, the police need to present at the time of the crime and see it being committed to make a lawful arrest (National Alliance on Mental Illness, 2017). Usually, in this case, the police would opt for assigning Joe a citation to appear in court rather than making an arrest (National Alliance on Mental Illness, 2017).

After Joe is taken to the police station, he will have to go through an administrative procedure, booking, during which he will be fingerprinted and photographed as a record of his arrest (National Alliance on Mental Illness, 2017). Once he arrives at the police station, Joe's family should be notified immediately and, in the case of a misdemeanor charge, disclose to the police of psychiatric history and circumstance that could have impacted the Joe's behavior (National Alliance on Mental Illness, 2017). However, in the case of a felony charge, this should be done cautiously and after discussion with an attorney, because it could make the police take the case more seriously. If the best option is to disclose this information, Joe's family should use it to persuade the station chief to transfer the offender to a mental health facility to receive treatment (National Alliance on Mental Illness, 2017). In addition, Joe should be prevented from being interrogated without a lawyer present and interrogation should cease as soon as Joe asks for a lawyer (National Alliance on Mental Illness, 2017). However, in the case that the interrogation does happen, if Joe is not warned of his Miranda rights, the police cannot use any statements made by the offender later in trial (National Alliance on Mental Illness, 2017). The police are likely to question Joe about the crime to obtain a confession or psychiatric history to use against potential later insanity defense to the crime (National Alliance on Mental Illness, 2017).

After Joe has been booked, both the police and a superior or member of the prosecutor's office will decide whether to file charges depending on different factors, like the probability that Joe will remain dangerous, the frequency at which Joe gets into trouble, and the quality of care Joe can receive if charged (National Alliance on Mental Illness, 2017). At this point, Joe's family may want to consider informing the prosecutor of the nature and effects of Joe's brain injury (National Alliance on Mental Illness, 2017). If police initially arrested Joe for a felony, it is

likely for Joe to be charged with aggravated assault and will not be released (National Alliance on Mental Illness, 2017). However, they might also decide to reduce the felony charge to a lesser felony charge or a misdemeanor (National Alliance on Mental Illness, 2017). They also have the option to put Joe on probation or drop the charges entirely in favor of jail diversion, a procedure in which a person with mental illness agrees to participate in treatment or some other activity in exchange for being charged (National Alliance on Mental Illness, 2017). Jail diversion could have potentially been an option for Joe twice during the proceeding, both earlier when the police could have delivered Joe to the mental health facility straight from the scene of the crime or now before pressing charges. Jail diversion is different than probation, a suspended sentence, that would occur if the judge set a sentence for Joe that includes jail time but allows Joe to stay out of jail during the period of the sentence given he stays out of trouble (National Alliance on Mental Illness, 2017). Probation is different from jail diversion in how Joe still has the possibility of carrying out the rest of his sentence in jail (National Alliance on Mental Illness, 2017). Jail diversion is a beneficial and preferred alternative to all the other options for Joe, because it allows Joe to avoid a history of criminal charges for crimes that were only an effect of Joe's TBI or mental illness, although this method is only possible in the case of a non-violent misdemeanor or minor felony. Another option that would allow Joe to avoid incarceration and assure treatment is plea bargaining, a process that involves negotiations between the prosecution and the defense to reach a mutually agreed disposition (National Alliance on Mental Illness, 2017). Plea bargaining ends up resolving most criminal cases and can occur at any point in a criminal case (National Alliance on Mental Illness, 2017).

However, if the police decide to press charges against Joe, Joe will become a defendant and a complaint will be filed to the magistrate's court detailing the charges (National Alliance on Mental Illness, 2017). If the magistrate also decides there is sufficient legal basis to proceed, Joe must be presented before the magistrate, a procedure known as "first appearance" or "arraignment," without unnecessary delay (National Alliance on Mental Illness, 2017). At his first appearance, the magistrate will first make sure he is the one who committed the crime,

inform him of his rights, appoint him an attorney if he cannot afford one and if his charge is likely to result in incarceration, otherwise known as imprisonment, and set his bail or order pre-trial detention (National Alliance on Mental Illness, 2017). Bail is a collateral, in the form of money, that the court holds that allows the defendant to remain free while their case is pending (National Alliance on Mental Illness, 2017). It serves as a substitute for pre-trial detention, which the magistrate will order only if he believes Joe won't show up for trial (National Alliance on Mental Illness, 2017). It is extremely beneficial for a lawyer to be present at the hearing to serve as an effective advocate who can explain to the court the circumstances to justify a lower bail (National Alliance on Mental Illness, 2017). Discussing the issue of illness resulting from traumatic brain injury may or may not be beneficial at this point based on the judge's reaction and sensitivity to such issues (National Alliance on Mental Illness, 2017).

The due process clause of the Constitution states that no defendant can stand trial and be sentenced if "because of a mental disease or defect he cannot understand the nature of the proceedings against him or cannot assist his lawyer in preparing his defense" (National Alliance on Mental Illness, 2017). If Joe is found incompetent but is deemed able to regain competence, he will be treated at a mental health treatment facility until he does so (National Alliance on Mental Illness, 2017). However, if Joe deemed unable to regain competence, the charges will be dropped but he will be civilly committed (National Alliance on Mental Illness, 2017). In summary, this clause makes it so that all incompetent defendants will have received jail diversion earlier on or treatment during this phase (National Alliance on Mental Illness, 2017).

In a misdemeanor case, the actual trial is held at this point in the proceedings. In a felony case, the next step is the preliminary hearing, during which the magistrate judge will determine if there is sufficient evidence to continue with the prosecution, or the grand jury, which is similar to a preliminary hearing except a group of civilians are brought in and review the case instead (National Alliance on Mental Illness, 2017). If the charges survive the preliminary hearing or the grand jury, an indictment, containing the original charges with modifications, will be filed and Joe will have to make a plea of not guilty, no contest, or guilty, a process known as an

arraignment of the indictment (National Alliance on Mental Illness, 2017). If Joe pleads no contest, it means he does not admit guilty for the crime but will allow the court to determine the punishment (National Alliance on Mental Illness, 2017). Most people who plead no contest instead of guilty does so to avoid being sued for confessing to the crime (National Alliance on Mental Illness, 2017).

After Joe pleads guilty, no contest, or not guilty, he will have a trial, during which the prosecution and the defense get to make their arguments regarding events leading to the crime before a judge and/or a jury of six to twelve civilians (National Alliance on Mental Illness, 2017). In Joe's case, he may or may not testify in court, per advice of his lawyer, and may benefit from use of the insanity defense (National Alliance on Mental Illness, 2017). However, most individuals with mental illness or traumatic brain injury, even if they were not in full control when they committed the crime, will not qualify from the insanity defense, which is used only if the offender had no idea what they were doing at the time. The insanity defense wouldn't be of much use in these cases and are usually reserved for more serious felonies, as Joe would have probably went through jail diversion if he was charged for a misdemeanor and could have qualified for the insanity defense (National Alliance on Mental Illness, 2017).In most states, a verdict at a trial must be unanimous for the defendant to be pronounced guilty or not guilty (National Alliance on Mental Illness, 2017). If an unanimous verdict is reached, double jeopardy will ensure that Joe can never be tried again on the same charges (National Alliance on Mental Illness, 2017). If an unanimous verdict cannot be reached, the prosecution can re-try the case before a new jury (National Alliance on Mental Illness, 2017). Misdemeanor cases usually don't require juries and will be tried before only a magistrate judge (National Alliance on Mental Illness, 2017).

The insanity defense, if applied, will mean that although Joe did commit the crime that he is being charged for, he is not guilty of the crime because he is insane, meaning that he "lacked sufficient capacity to appreciate the criminality of his or her acts, or to conform his or her actions to the requirements of law because of a mental disease or defect" (National Alliance on Mental

Illness, 2017). Most states, aside from Kansas, Montana, Idaho, and Utah, allow for the insanity defense, though it is mostly only used for serious felonies (National Alliance on Mental Illness, 2017). Once the defense raises the issue of insanity, the prosecution must prove that the defendant was sane when committing the crime (National Alliance on Mental Illness, 2017). The state is then also required to provide the defendant with a psychiatrist, who usually are concerned professionals with balanced opinions but may also lean towards the opinion that will ensure them future assignments (National Alliance on Mental Illness, 2017). If the prosecution finds Joe not guilty by reason of insanity, then he will be committed to a forensic facility, containing state facilities and community services for individuals with mental illness, until he regains sanity or becomes safe to the community (National Alliance on Mental Illness, 2017). The prosecution can also find Joe guilty but mentally ill, a deviation from guilty or not guilty, and apply the same punishments corresponding to a traditional guilty verdict but also give Joe treatment for mental illness during incarceration (National Alliance on Mental Illness, 2017). This verdict will recognize the impact of brain injury and mental illness on Joe's decision to commit the crime and ensure that Joe will not be prematurely released into the community, which is more likely to occur if Joe is found not guilty by reason of insanity (National Alliance on Mental Illness, 2017).

Joe is more likely to qualify for mitigating circumstances, which is different from "not guilty" by reason of insanity in how it will not excuse Joe from the crime but may result in Joe being convicted of a lesser crime (Cornell Law School). One such mitigating circumstance is the "diminished capacity" plea, which was established to excuse people who are too mentally impaired to intend to cause a death, and therefore must have caused the death recklessly and without a guilty mind (Cornell Law School). Other examples of mitigating circumstances involves age or history of the defendant, "heat of passion," remorse, addiction, etc…(Cornell Law School).

If Joe is pronounced guilty, he will be sentenced by a judge who has the option of imposing imprisonment or probation (National Alliance on Mental Illness, 2017). The sentence is usually determined by the statue corresponding for Joe's particular crime and the pre-sentence

report written by a probation officer (National Alliance on Mental Illness, 2017). Joe's family should contact the probation officer beforehand to provide information on Joe's brain injury and mental illness, and treatment requirements, for the report (National Alliance on Mental Illness, 2017). For a felony case, incarceration is a likely outcome, however, for a misdemeanor case, it is likely Joe will receive a suspended sentence with a period of probation and psychiatric medical treatment (National Alliance on Mental Illness, 2017). Lastly, if Joe is convicted, he has the right to appeal a conviction and have a higher court review the trial court judge's decision to see if the judge had made a legal error during the trial (National Alliance on Mental Illness, 2017).

The due process clause and the Eighth Amendment of the Constitution guarantees every incarcerated individual with a "serious medical need" the right to appropriate medical treatment (National Alliance on Mental Illness, 2017). Therefore, it is important for Joe's family to demonstrate that Joe has a "serious medical need" so that, in case of incarceration, he will be treated in prison (National Alliance on Mental Illness, 2017). This is more difficult than it should be, since this decision usually falls on the medical professionals involved in the case who only start observing the incarcerated in jail or prison (National Alliance on Mental Illness, 2017). The jail or prison setting is not conducive towards accurate diagnosis of inmates with mental illness, since inmates may significantly change their behavior and lose usual personality characteristics, like withdrawing into themselves instead acting in a disrupt manner, that would point to mental illness (National Alliance on Mental Illness, 2017). Oftentimes, individuals in need of treatment escape the radar for years (National Alliance on Mental Illness, 2017). In addition, this clause only entails treatment to alleviate the more severe symptoms of mental illness and doesn't include treatment to prevent recurrence of illness and rehabilitative services necessary for inmates to acclimate back to the community after being released (National Alliance on Mental Illness, 2017). Therefore, it is important for the family members to communicate all information on their family member's mental illness both pre-trial to the sheriff and jail administrator and post-trial to the prison warden and chief medical officer, but on the advice of their attorney (National Alliance on Mental Illness, 2017).

If Joe had committed a crime against the members of his family due to delusion or some sort of brain alteration, for example, if he had tried to kill his mother because he believed she was trying to kill his father, the family should first call the police and then try to convince them to transport Joe to a mental health facility instead of arresting Joe (National Alliance on Mental Illness, 2017). However, this option may not work because emergency treatment only lasts one to three days and can only be extended through civil commitment, which is not always accessible or available (National Alliance on Mental Illness, 2017). Emergency treatment may also not treat but only confine Joe for a couple days before releasing him (National Alliance on Mental Illness, 2017). The other option, to press charges against Joe, would follow the procedures outlined above but would be a little different because the prosecutor would be unlikely to proceed with the case unless supported by Joe's family or if Joe is a danger to society (National Alliance on Mental Illness, 2017). Joe's family could suggest the jail diversion program to the prosecutor or recommend a sentence that would include mandatory treatment at a mental health facility (National Alliance on Mental Illness, 2017).

Role of Neuroimaging and Evidentiary Standards in Mitigation

Techniques have been created and improved upon enormously in the recent decades. Individual neurons can be studied, optogenetics can be used to monitor the activities of neurons, neural networks and connections can be mapped, and high-resolution brain imaging can be developed. Due to such advances, we now have a more thorough understanding of mental disease. Former President Obama had launched the Brain Research through Advancing Innovative Neurotechnology, BRAIN, initiative to "accelerate the development and application of new technologies that will enable researchers to produce dynamic pictures of the brain that show how individual brain cells and complex neural circuits interact" (BRAIN). This project focuses on technology development to determine the roles of varying brain cell types, generate circuit diagrams to see the brain in action, monitor neural activity on a large-scale, use interventional tools to determine how brain activities determine behavior, and to use these

technologies to determine how neural activity is translated into an individual's perception, actions, emotions, and cognition.

As technology progresses, scientific evidence will play an increasingly greater role when it comes to indictment of criminals, especially when determining if the defendant can qualify for the "diminished capacity" plea. Cognitive neuroscientists have located brain regions for empathy, impulse control, and moral decision-making (Edersheim, Brendel & Price, 2012). Neuroimaging can shed light on the "*mens rea*" part of criminal offense for offenders with traumatic brain injury by unveiling brain abnormalities, which can mitigate the severity of sentence.

Legal scholars have described four different mental states to go with the "*mens rea"* clause: purposefulness, knowledge, recklessness, and negligence (Edersheim, Brendel & Price, 2012). Purposefulness is a combination of voluntary action and intent and is exhibited by an individual who acts with a conscious purpose to cause a specific result (Edersheim, Brendel, & Price, 2012). Knowledge is awareness that one's actions will cause that specific result (Edersheim, Brendel, & Price, 2012). Recklessness is a disregard for "substantial and unjustifiable risk" (Edersheim, Brendel, & Price, 2012). Lastly, negligence is the creation of the risk despite knowing the consequences (Edersheim, Brendel, & Price, 2012). Neuroimaging can help with clarifying the meaning behind the first two mindsets, purposefulness and knowledge, by identifying deficits in specific brain regions associated with elevated aggression and violence (Edersheim, Brendel, & Price, 2012). Deficits in these brain regions can substantiate the claim that the defendant could not have acted with purpose or knowingly while committing the criminal act (Edersheim, Brendel, & Price, 2012). However, brain imaging is not completely objective, given how casual links have not yet been established between visible abnormalities and behavior and how abnormal brain activity does not necessarily imply brain impairment (Edersheim, Brende, & Price, 2012).

Since neuroscience cannot definitively determine the causality between brain dysfunction and mental culpability, and thus cannot absolve an offender of criminal responsibility with

absolute certainty, neuroscience should be used more in the "sentencing phase" rather than the "guilt phase" to help reduce an offender's sentence. One way neuroimaging can affect sentencing is through the diminished capacity defense. This plea operates as a lesser form of legal insanity that allows the defendant to introduce evidence of mental impairment at the time (s)he committed the crime to lessen the criminal responsibility on the defendant. (Edersheim, Brendel, & Price, 2012). Psychiatric testimony that signifies the defendant was not capable of and did not premediate and deliberately commit the crime is necessary to convince the court that the defendant did not have specific *mens rea* (Edersheim, Brendel, & Price, 2012). In theory, if a defendant was able to empirically demonstrate he lacked the ability to formulate intentions or premediate his actions by introducing neuroimaging data showing a deficit in a brain region responsible for this ability, he might be able to show that he lacked *mens rea* for the crime (Edersheim, Brendal, & Price, 2012). So far, defendants have had little success in convincing judges and juries of brain abnormalities with neuroimaging that excuse them from their crime, but these claims have gotten more frequent over the past few years (Edersheim, Brenda, & Price, 2012).

Neuroimaging evidence can also play a role in presentation of mitigation evidence, which involves diminished criminal responsibility as opposed to diminished capacity, that fall into three categories: future threat to society, offender culpability, and character (Edersheim, Brenda, & Price, 2012). Mitigating factors are different from *mens rea* defenses in how there is no requirement for the evidence to have a mental disease or defect (Edersheim, Brenda, & Price, 2012). Juries tend to take into consideration mitigating facts and circumstances that show the defendant had impaired mental capacity while committing the crime (Edersheim, Brenda, & Price, 2012). One study indicated that juries were much more likely to excuse a defendant by reason of insanity if neuroimaging results had been presented, and it is also required by the Constitution that jurors heavily take into consideration a list of aggravating and mitigating factors when considering the death penalty sentence (Edersheim, Brenda, & Price, 2012). Neuroimaging can be a double-edged sword when presented as one of these factors, because it

may constitute an aggravating, as opposed to a mitigating, factor (Edersheim, Brenda, & Price, 2012).

There have been some notable cases of trials involving defendants with traumatic brain injuries. The first of which, *State v. Reid,* convicted and sentenced Reid to death for multiple counts of first-degree murder (Edersheim, Brenda, & Price, 2012). Defense experts presented evidence in support of various mitigating factors, including a rough childhood, four severe head injuries with a resulting psychotic disorder, and diagnosis with a schizoaffective disorder (Edersheim, Brenda, & Price, 2012). MRI and PET scan were also presented and revealed atrophy of Reid's left temporal lobe probably resulting from previous childhood head injury (Edersheim, Brenda, & Price, 2012). However, the court ultimately decided that aggravating factors, including future threat to society, outweighed the mitigating factors and still sentenced Reid to death (Edersheim, Brenda, & Price, 2012). In *Rogers v. State,* Glen Rogers was convicted of first-degree murder and sentenced to death (Edersheim, Brenda, & Price, 2012). During his first trial, the court had rejected his motion for a PET scan (Edersheim, Brenda, & Price, 2012). After his defense experts brought up his history of head trauma, skull fracture, and subsequent seizure disorder, the trial allowed for presentation of an MRI, but it did not end up revealing brain abnormality (Edersheim, Brenda, & Price, 2012). Finally, in *Ex Parte Simpson,* Simpson was convicted of robbery and murder of a retired school teacher and sentenced to death (Edersheim, Brenda, & Price, 2012). During trial, his defense expert provided evidence of previously diagnosed borderline intellectual functioning and antisocial personality disorder, a MRI showing two subdural hematomas, an EEG revealing non-specific generalized slowing of mental capacity, and gave the opinion that his brain injuries had caused poor judgment, decision-making skills, inability to learn from past mistakes, and inability to control his frustration and emotions (Edersheim, Brenda, & Price, 2012).

Standards to divide responsible from the non-responsible

After discussing the clinical diagnostic criteria of TBI, downstream symptoms that could lead to violent tendencies, and criminal justice procedure, it is finally time to determine the different factors that separate defendants with history of TBI into those who are criminally responsible and those who are not.

It is first critical to establish that the offender does indeed have a history of TBI. If there are hospital records, for a head injury to be established as TBI, the offender must have had any of the following: change in mental state during time of injury (confusion, dull mental skills, etc…), post-traumatic amnesia, decreased or loss of consciousness, ensuing neurological deficits, and/or other evidence of brain pathology. However, since many individuals are not hospitalized, unaware, or have no record of previous TBI, this may be difficult. If this is the case, the attorney may have to rely on second-hand accounts from friends and family to determine if the offender had previously suffered from a traumatic head injury.

Then, the severity of the offense needs to be considered. In a misdemeanor case, there are several points in the criminal justice procedure for intervention on behalf of those with altered mental states before the situation escalates to trial. The offender could undergo pre-trial jail diversion and the family of the offender should try to persuade the authorities to exercise this option. However, in many of the case studies, the subjects had committed serious felonies, so there was no way around going to trial. Although many of these subjects will be pronounced guilty and sentenced for some time, which is the safest choice for not only them but the community, neuroimaging evidence should be procured in trial by a defense expert in the hopes of enabling the subject to receive a reduced sentence or charge. A clear correlation between the defendant's brain injury and subsequent personality change, and/or other symptoms, must be established, and presented as a mitigating factor or evidence of diminished capacity.

I believe the mitigating factor that should be taken the most into account, in cases of defendants with TBI, is evidence of abnormal brain pathology. Previously, there has been a lack of neuroimaging evidence in courts, and it is also likely the evidence may not be well-received, due to how there is still no clear casual relationship established between TBI and offending

behavior that can be pointed out from a brain scan. However, modern brain science has produced more advanced neuroimaging evidence and with it, more questions such as, what were the intentions of the defendant? and did the defendant pre-meditate the murder? can be answered. Ultimately, the goal of presenting neuroimaging evidence is to persuade the court that the defendant did not commit the guilty act while possessing a guilty mind, so they might not hold the defendant criminally responsible for his crime. In a lot of cases with defendant with TBI, the defendant did not intend to kill his or her victim but instead lost control of themselves during the act. These questions should be answered and analyzed to varying degrees, either by using neuroimaging or neurological assessment/evidence, in the hopes of deeming the defendant with impaired impulse control or neurological deficits. Hopefully, in the future, these technological developments can make it possible for brain imaging to provide objective, concrete, and reliable evidence of potential answers to these questions. Other mitigating factors that should be considered, but to a lesser degree, should be no criminal history, environmental circumstances that indicate the offense was out-of-character for the defendant, evidence of other mental illnesses, remorse for having committed the crime, and possibility of rehabilitation.

Aggravating factors is something else to take into consideration. In many cases, defendants with history of TBI had been sentenced to death or imprisoned due to presence of too many aggravating factors. Since TBI, particularly when advanced to chronic traumatic encepathology, is uncurable, oftentimes it is necessary for defendants to be imprisoned due to lack of capacity to control their symptoms, like aggression that led them to their criminal tendencies. However, in other cases, proper therapy and medication in a mental institution and in prison might help in reducing severity of symptoms and increasing possibility of later assimilation into society.

Application to Case Studies

a. Charles Whitman

The amygdala aids in the learning of appropriate responses to stimuli that are threatening and fear-arousing, and helps the brain remember details of fearful situations so these situations can be avoided in the future (Social Psychology). However, although its primary function is to help individuals perceive and respond to danger, it also is connected to portions of the brain, such as the prefrontal cortex, that functions in the inhibition of aggression (Social Psychology). The tumor Charles Whitman had, though impacting the amygdala, was in the temporal lobe and obstructed pathways extending from the amygdala to the thalamus and hypothalamus, meaning that these impaired pathways, especially the one extending from his amygdala to his hypothalamus, could have contributed to Whitman's lack of fear response to his actions and his compulsive need to shoot people from the tower (Wallenfeldt, 2018). However, it is still not clear if how in control Whitman was of his actions. Some people believe Whitman's violence was due to anger towards his family, given his history of child abuse, and discontent towards his situation (The Daily Texan). So, to what extent is Whitman culpable? Should the blame be placed on the perpetrator or biology? More broadly, how should the criminal justice system approach incriminating individuals who have damaged brain areas?

For Whitman case, it appears the tumor had played a big role in motivating Whitman's actions and thus the blame cannot be solely attributed to Whitman. Neuroimaging evidence has provided concrete evidence of the existence and location of the tumor, but not how the tumor could have affected his actions. However, since the tumor was specifically affecting the amygdala, the fact that his actions stemming from the tumors perfectly aligned with actions resulting from amygdala defect is too big a coincidence to ignore. Therefore, this evidence should have convinced the judges, if Whitman did go to trial, that he wasn't a cold-blooded murderer and was heavily influenced by the tumor. Other mitigating factors include his actions prior to the shooting. Whitman had gone to the UT Mental Health Center and told everything to a therapist- his desire to commit murder, and his plan to shoot people from the UT tower. He obviously was aware of what he wanted to do was wrong and was trying to right the wrong by getting help. He also had no committed any crimes prior. There may have been environmental

and societal factors that may have influenced his decision-making skills as well. Whitman's father had abused him and his mother as a child and Whitman might have had PTSD or some mental illness developed from his time in the Marines. All of these are mitigating factors that need to be considered.

Some aggravating factors that should be considered is premeditation of the murder, Whitman's violent personality, and how the presence of his tumor should not entirely excuse him from the crime, since there are many people with tumors who do not end up committing mass murder. It is also uncertain how big of a role the tumor played in his actions. Whitman had previously been employed in the military, indicating that he was fine with exposure to violence, and his actions might have bene facilitated through his life experiences as well. In addition, the line between brain trauma vs. character traits is murky. There is always a degree of uncertainty when it comes to diagnosing someone with TBI and making their symptoms clinically relevant. If the offender is prone to impulsive behavior, is it because they have TBI or simply because they enjoy behaving impulsively?

Ultimately, due to lack of previous offense, evidence of brain trauma, as well as severity of the offense, it is fair for the court to sentence Whitman with first-degree murder, given the extensive premeditation that was put into it, but also with treatment and tumor dissection surgery. And, since part of the blame can be attributed to the tumor, there should be possibility of a re-trial after the surgery if Whitman improves.

b. Aaron Hernandez

Some mitigating factors that should have been considered in the case of Aaron Hernandez include, first and foremost, neuroimaging evidence of CTE that was only provided after his death. Considering how he had been an active football player for most of his life, the court should have ordered brain scans before sentencing him. Other factors include his abusive childhood, mental health issues, drug and alcohol abuse, etc…

Some aggravating factors that should be considered is his high likelihood of recidivism, since Hernandez had been actively involved in violent confrontations and bar shootings before being arrested, his lack of remorse, seen from his plea of "not guilty" and attempted cover-up after the crime, and the knowledge that most people with CTE do not end up committing murder. Although Hernandez was by no means insane or unaware of his actions, the damage to his brain, along with his past experiences with domestic and drug abuse, along with the environment he was raised in, contributed to his aggression and eventual criminal actions. Hernandez did not premediate the murder to the extent Charles Whitman did, but he was less restrained in his aggression and criminal behavior leading up to the homicide. Hernandez also seems less self-aware than Whitman, who clearly knew his actions were wrong, and justified his actions by blaming others for provoking him or going against him in some way. Additionally, there is no treatment for CTE, only strategies for detection and management, and the severity of Hernandez's CTE was beyond repair.

Aaron Hernandez was found guilty of murder of Odin Lloyd in the first degree and sentenced to a life in prison without possibility of parole (Boston Globe). He began his sentence in a maximum-security intake facility and was then transferred to a similar facility until his death (Boston Globe). He was kept in a segregated unit with others who were violent and mentally ill, in the first facility and was not allowed to move out of segregation until he switched to the second facility. In addition to having to endure grim conditions, Hernandez was not treated well and was constantly taunted by guards and was still taking drugs and tortured over his homosexuality (Boston Globe). He did not receive psychiatric treatment or any type of therapy while in prison, and as a result, his conditions didn't improve. Although Hernandez was by no means insane or unaware of his actions, the damage to his brain, along with his past experiences with domestic and drug abuse along with the environment he was raised in, contributed to his aggression and eventual criminal actions. Hernandez did not premediate the murder to the extent Charles Whitman did, but Hernandez was less restrained in his aggression and criminal behavior leading up to the homicide. He also seems less self-aware than Whitman, who clearly knew his

actions were wrong, and justified his actions by blaming others for provoking him or going against him in some way. There is no treatment for CTE, only strategies for detection and management, and the severity of Hernandez's CTE was beyond repair. Although the jury rightfully imprisoned Hernandez, who would have remained a threat to the community, they should have also considered his mental illness and brain injuries and given him psychiatric treatment during imprisonment.

CONCLUSION

"And a man who injures his countryman- as he has done, so it shall be done to him [namely], fracture for a fracture, eye for eye, tooth for tooth. Just as he has injured a person, so it shall be done to him." (Lev. 24:19-21)

"An eye for an eye," otherwise known as the law of retaliation, makes it fair for someone to hurt another to a similar degree as they were hurt by the other person. This law authorizes retaliation and it was first noted in the Code of Hammurabi and upheld in the Babylonian law. Though it is unfair to commit a crime without punishment, an eye for an eye is not a just punishment. The crime commuted is oftentimes extremely complex and involving several factors, one of which being biological, and resolving it justly and fully requires more than just blind equal retribution. There are just too many exceptions to this law, too many cases during which it wouldn't provide equitable results. There is no apparent trend in how neuroscientific evidence, due to its inability to determine the casual relationship between brain trauma and criminality, can be used to mitigate an offender's sentence. However, it is clear that such evidence only bears weight, and only as a mitigating factor, in the presence of other favorable circumstances. In other words, neuroscientific evidence is nothing more than one mitigating factor among many and will not be seen as significant unless there are other factors for the defendant playing into a more lenient sentence. However, as the field of Neurolaw progresses, new insights into an offender's mental state and criminal responsibility can be discovered. The large intersection between criminal justice and health policy will make it essential for the justice system to keep adapting to better assess and incarcerate those afflicted with biological brain issues.

Much of this thesis was spent deliberating the question, "to what extent is the offender criminally responsible?" In retrospect, this question does not aid in the facilitation of therapeutic and medical treatment for those who are indicted and afflicted. Society should spend more time

in establishing changes to the system and correction facilities that could reverse the effects of TBI while also separating the dangerous from society. The biggest issue with the criminal justice system is lack of incorporation of mental health treatment into sentencing, when many defendants with TBI desperately need treatment in prisons.

Although we know much more about consequences of brain injury than we did decades ago, future neuroscience discoveries will continue to alter and change the way education and legal systems operate. It will deepen our knowledge of why people act the way they do and how to best treat and support them. It will also help the legal system deal better with violent criminals, move behavior towards socially desirable directions, and produce just trial results.

It is pertinent to keep seeking better solutions while remaining open-minded in the face of concepts such as “free will.” Even though correlation does not equal causation, these advancements can provide us with more reliable sources of evidence of brain trauma, which can lead to better medication and combative strategies. The future should be spent on advancing this trend and becoming more open-minded that maybe the brain, in some cases, is to blame.

BIOGRAPHY

Andi Liang was born in Beijing, China on July 10th, 1997, raised in Troy, Michigan, before moving with her family to Sugar Land, Texas in 2011. She enrolled in the Plan II Honors Program and in the College of Natural Sciences at the University of Texas at Austin in 2015 and studied for four years before graduating with a B.S. in Human Biology and a B.A. in Plan II Honors in 2019. During college, she pursued medicine by interning with the Health Leadership Apprentice Program at Dell Medical School and finding employment at medical clinics, such as the NeuroAustin Neurology Clinic. She plans on attending McGovern Medical School in the fall, where she will continue pursuing her passion for medicine and life.

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